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A TEXT-BOOK

OF THE

ACTICE OF MEDICINE

BY

DR. HERMANN EICHHORST

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Clinic in the University of Zurich

AUTHORIZED TRANSLATION FROM THE GERMAN

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VOLUME I

PHILADELPHIA AND LONDON
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EDITOR'S NOTE.

those who read German no introduction to Eichhorst's
e of Medicine will be necessary ; and it is hoped that this
tion may prove not less useful than the original, and as
. The text will be found at once concise and compre-
, but additions and annotations have been made where it
they might be serviceable. The book differs somewhat
ost other works on the practice of medicine in containing,
other things, appropriate sections devoted to a considera-
Diseases of the Skin, the Venereal Diseases, Impo-
ad Sterility in the Male, and Spermatorrhea. A number
trations have been added, in part replacing, in part sup-
ting, those in the original. The book has been written
ly to meet the needs of the student, but it should prove
lso to the busy practitioner. For convenience of manipu-
t has been deemed wise to present the work in two
s.

AUGUSTUS A. ESHNER.

ADELPHIA, March, 1901.

PREFACE.

impulse to write a short Text-book of Special Pathology
erapeutics has not emanated from myself, but from my
For a number of years the latter have repeatedly impor-
e, and all references to existing text-books written by me
vain. Recently the demand has been made also from
quarters, and I have therefore finally accepted it as a duty
nd to a request pressed with such persistence.

governing principle in the preparation of this work was
de the student with a book that should afford him a ready
able source of information with regard to the principal,
cially to the most important, manifestations for the recog-
diseases. As the essential aim of the physician will ever
re of disease, earnest consideration has been given to the
f treatment.

rally, this text-book can deal only with the established
the practice of medicine. A certain degree of conciseness
sion and in description also was demanded, and especially
onnection considerable difficulty was encountered in the
gorously to separate the essential from the unessential.
far I have been successful in this must be left to the
of the reader.

who are better acquainted with the manifestations of
nedicine will find the necessary elaboration in my *Hand-
Special Pathology and Therapeutics.*

ll be gratified to know that the present volume will real-
xpectations of those for whom primarily it was written.

HERMANN EICHHORST.

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[illegible] . . . . [illegible]

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PART I.

DISEASES OF THE CIRCULATORY ORGANS.

I. DISEASES OF THE MYOCARDIUM.

THE termination in cases of heart-disease of all kinds depends in the majority upon the functional capability of the myocardium, and death occurs in most cases amid manifestations of overwhelming weakness of the heart-muscle. Easy as it is to diagnose conditions of weakness or insufficiency of the myocardium, it is equally impossible, as a rule, to recognize during life the anatomic alterations that have taken place in the heart-muscle itself in every individual case. The anatomic diagnosis of disease of the myocardium will not reach beyond a certain degree of probability even under favorable conditions; and unfortunately often this degree is not obtained even by experienced, observant, and careful diagnosticians.

The diagnostic scope of diseases of the myocardium is thus most circumscribed, and virtually includes only three conditions: First, that of *weakness or insufficiency of the myocardium,* already mentioned; second, *enlargement or dilatation of the heart;* and third, *increase in volume, or hypertrophy, of the myocardium.*

WEAKNESS OF THE MYOCARDIUM (CARDIAC INSUFFICIENCY).

Symptoms and Diagnosis.—The symptoms of enfeeblement of the myocardium are partly *local,* partly *general,* or in many instances of *mixed* character.

The **local,** or strictly **cardiac,** manifestations usually attend the onset. The action of the heart is generally accelerated inordinately, and at the same time it is usually irregular. While at first these disturbances perhaps only follow physical and mental exertion, large meals, or the use of stimulants, later they appear also during complete rest and a careful mode of life, lose their paroxysmal character, and remain permanent. The patient often complains of a sense of distressing palpitation of the heart, at times also of a feeling of tension and pressure, less commonly of a painful sensation in the region of the heart, suffers from shortness of breath, and speaks interruptedly and at short intervals—*staccato speech.*

The *apex-beat* of the heart appears feeble or is not palpable. The *sounds* of the heart are faint, and often the first (systolic) sound is suggestive of a murmur. Not rarely *gallop-rhythm* is present, the systolic sound of the heart being preceded by a beat, the accentuation, however, falling upon the first sound itself. As a rule, increase in the area of *cardiac percussion-dulness* to the right and left, in consequence of dilatation of the heart, is demonstrable.

The disturbances of cardiac action can be admirably studied in the *radial pulse.* This attains unusually great frequency, exhibits very unequal waves, and is irregular and frequently also intermittent, in consequence of which some pulse-beats become inappreciable, because the force of the heart is not sufficient to send a palpable wave of blood into the radial artery with every contraction of the heart-muscle. As can be understood, the features of a frequent, irregular, unequal, and intermittent pulse will appear also in the *sphygmographic tracing* (Fig. 1).

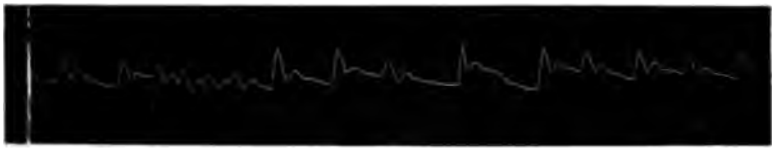


FIG. 1.—Sphygmographic tracing of a frequent, irregular, unequal, and intermittent pulse attending cardiac weakness in consequence of a valvular lesion (personal observation, Zurich clinic).

Only rarely are the consequences of weakness of the heart-muscle confined to the local or cardiac alterations described, and, as a rule, **general disturbances** or **manifestations of stasis** appear at the same time or more frequently somewhat later.

If the functional activity of the right ventricle is impaired to such a degree that it is no longer capable of sending all of its blood into the pulmonary artery with each systole, and a certain amount of blood will thus remain in the ventricle after each systole, the flow of blood from the right auricle will first be interfered with, and at the same time also that from the superior and inferior venæ cavæ to the heart, and the condition of general venous stasis becomes established. [illegible] appreciable earlier and in greater degree in the [illegible] inferior vena cava, as here the blood [illegible] force of gravity.

If with weak[illegible] ventricle is especially involved, [illegible] the same as that described. [illegible] left ventricle and in the [illegible] the pulmonary

e pulmonary capillaries, the pulmonary arteries, and from
turn to the right ventricle, the right auricle, and the
væ, so that the consequences of conditions of enfeeblement
ight and of the left heart are the same.

ng the earliest indications of general venous stasis is *edema*
in. At first this appears on the inner aspect of the thigh
ut the ankles. At this time, also, it frequently recedes
the night, thus in the horizontal position of the body.
owever, the cutaneous edema becomes persistent, increas-
tent, and advancing with progressive stasis to the geni-
the posterior, lateral, and inferior portions of the anterior
the abdominal wall, to the posterior and lateral aspect
est, to the arms, and even to the face. In patients accus-
lie especially on one side of the body, the edema of the
lso more marked upon that side.

ma of the skin has existed for a long time or if it recur persist-
hickening of the cutis takes place, especially in the legs, and
is of greater or less degree develops.

lition to edema, general venous stasis gives rise to *cyano-*
skin and mucous membranes. This is due to the fact that
rdation of the blood-current the blood gives off an un-
rge amount of oxygen to the tissues, and at the same
omes overladen with carbon dioxid from them. Lips,
ip of the nose, ears, elbows, dorsa of the hands, knees,
of the feet are the parts in which cyanosis of the skin
arliest and most distinctly, for in these places the skin
d at the same time rich in blood-vessels.

cheeks and the nose *dilatation of cutaneous veins* frequently takes
hich ultimately chronic inflammatory hyperplasia of the skin
lly be superadded, so-called *acne rosacea.*

pation *reduction of the temperature of the skin* is generally
and this may be explained by the fact that with retar-
the blood-current unusually active cooling of the blood
lace.

ction to the flow of blood from the renal veins into
r vena cava gives rise to manifestations of *hypostatic*
kidney and the *urine of stasis.* The urine is voided in
l amount (less than 1500 c.c.), and is characterized by
d (high, saturated) color and high specific gravity (above
exhibits an acid reaction, frequently contains small
albumin and hyaline tube-casts, and often on cooling
red, brick-dust-like (lateritious) sediment, which consists
of acid urates, and mainly of acid sodium urate. On
examination this appears in the form of irregularly
granules. Increase and diminution in venous stasis
ely reflected in the state of the urine.

Naturally stasis of the blood in the inferior vena cava will also interfere with the discharge of blood from the hepatic veins, and will consequently give rise to the *hypostatic* (*cyanotic*) *liver*. Under such conditions the liver increases in size, so that its lower border may reach to the level of the umbilicus or still lower. The organ is sensitive on palpation and unusually hard. The patients also complain frequently of a sense of tension or pressure, or even of pain, as well as of constriction in the region of the liver. Not rarely hypostatic catarrh of the biliary passages develops, with the appearance of jaundice (icterus), which is frequently confined to the ocular conjunctivæ, but at times involves also the general cutaneous integument. If cyanosis be present in addition to icterus, the skin frequently acquires a slight greenish tint, *icterus viridis*.

Venous stasis generally extends from the area of the hepatic veins to that of the portal vein, and accordingly symptoms of *stasis* referable to the *stomach*, the *intestine*, the *spleen*, and the *peritoneum* will appear. Stomach and bowel become the seat of hypostatic catarrh, characterized by loss of appetite, eructation, vomiting, flatulent distention in the epigastrium, and irregularity in evacuation of the bowels. Dilatation of the hemorrhoidal veins also not rarely takes place, and hemorrhoids form. The spleen becomes increased in size, and in still greater degree in consistency, and very considerable amounts of transudate often collect in the abdominal cavity (*ascites*).

The *thoracic viscera*, also, become involved in the manifestations of stasis. The patients often suffer from obstinate *hypostatic catarrh of the bronchi*. Not rarely *expectoration of blood* (*hemoptysis*) takes place, in consequence at times of rupture, at other times of embolic occlusion, of blood-vessels. On microscopic examination of the sputum cells diffusely stained with hemoglobin, or containing hemoglobin in the form of granules, needles, and even plates or tables, are frequently found. These are improperly designated *heart-failure cells*, although they occur also in association with other diseases, for instance fibrinous pneumonia, and they are believed to be in part alveolar epithelium and in part round cells. The pleural cavities become filled with transudate (*hydrothorax*). Similar changes take place in the pericardium, and *hydropericardium* is frequently observed.

Evidences of venous stasis are found also in the distribution of the *jugular veins*. These veins themselves are conspicuous on either side of the neck by reason of their unusual fulness and dilatation. The patients suffer from *hypostatic catarrh of the pharyngeal* and *nasal mucous membrane*, and at times from frequent *epistaxis*. Some complain of *tinnitus aurium* and *impairment of hearing*. At times the eyes protrude markedly, and in consequence of over-distention of the retrobulbar veins slight exophthalmos becomes appreciable. The blood-vessels of the

;iva appear dilated and tortuous, and often excessive lac-
cretion accumulates in the conjunctival sacs.

y patients complain of vertigo, introspectiveness, cerebral
;, disturbed sleep, and the like, which are related to venous
ıia of the brain.

duration and the course of weakness of the heart-muscle
in every instance upon the operative causes. If these can
kly removed, the manifestations of myocardial weakness
frequently recede within a short time. Naturally the
n must not have progressed too far, for then the danger
great that to heart-weakness will be added *paralysis of the*
Frequently the causes can only be temporarily removed or
ıed, and accordingly the signs of myocardial weakness
;ain and again. Many patients die in consequence of
hypostatic phenomena, for when accumulations of con-
: amounts of transudate have taken place in the peritoneal,
and pericardial cavities there is danger that heart and
ll be impeded in their movement to so high a degree that
ıtenance of life is impossible. Only rarely does death
:e in consequence of *unrestrainable pulmonary hemorrhage*.
: *edema of the skin* also is attended with danger to life, as
may rupture at various points, and thus permit of the
' edematous fluid. Infection with bacteria from the air
ı readily take place, and give rise to an erythematous or
tous inflammation of the skin, and general *septicemia* may
fatal termination. In some cases intercurrent accidents
e, and *pneumonia* especially not seldom occurs.

ic states of heart-weakness are among the most distress-
tions, and they may compel the patient to remain in bed
; time and may engender a large number of complaints.

ogy.—Weakness of the myocardium develops most fre-
hen **unusually large demands** are made upon the **functional**
! the heart-muscle, and with which the heart cannot per-
comply. Among such causative conditions are valvular
' the heart, chronic diseases of the respiratory organs,
ıns and dilatations (aneurysms) of the aorta or the pul-
tery, arteriosclerosis, and chronic diseases of the kidney.
over-exertion, for which of late excessive athletic exer-
untain-climbing, bicycling, rowing) especially afford
opportunity, and the ingestion of excessive amounts of
noderate use of beer) may also give rise to insufficiency
rt-muscle.

s of the myocardium itself (myocarditis, fat heart,
hinococci) also may readily be associated with weakness
ırt-muscle, because, in the development of these condi-
:ular structure of the heart, therefore functionally capable
:stroyed. At times the myocardium is involved through

extension of disease from adjacent structures, as, for instance, pericarditis, obliteration of the pericardium, and endocarditis.

Among the **toxic varieties of myocardial weakness** may be included those that arise after excessive use of alcohol, coffee, tobacco, and tea. In this group perhaps may be included also some infectious diseases, in which the toxins impair the integrity of the myocardium.

At times **general loss of bodily fluid** gives rise to weakness of the myocardium, as, for instance, in pulmonary tuberculosis, carcinoma, chronic diarrhea, and suppuration.

Occasionally weakness of the myocardium is dependent upon **alterations in the blood,** and it occurs in connection with chlorosis, leukemia, pseudoleukemia, pernicious anemia, or after the **loss of considerable amounts of blood.**

From the nature of the causative factors, weakness of the myocardium occurs but seldom in childhood. Conversely, a natural predisposition is present in advanced life—*senile weakness of the myocardium.*

Anatomic Alterations.—Weakness of the heart-muscle may cause death without any change in the myocardium whatever being discoverable, macroscopically or microscopically, at autopsy. In the majority of cases, it is true, the myocardium is marked at least by great flaccidity and dilatation, and often still other alterations in it will be found, such as have been suggested in the discussion of the etiology.

The **anatomic appearances of the hypostatic alterations** are readily recognizable. The *edema of the skin* remains demonstrable also in the cadaver. From the *serous cavities* (pleura, pericardium, peritoneum) large amounts of clear greenish-yellow transudate may often be evacuated. The *cavities of the heart*, especially the right ventricle and auricle, are greatly distended with blood and blood-clots, and on incision of the venae cavae such an abundance of blood escapes as to suggest the possibility of an increase in the amount having taken place. The *lungs* in long-standing cases present a brownish-red color, which, as microscopic examination proves, is caused by an abundant accumulation of blood-pigment. The lungs appear also remarkably dense, in consequence of increase in the interalveolar connective tissue, and the condition is therefore spoken of as *brown induration of the lungs.* The *mucous membrane of the bronchi* is conspicuous for its marked redness and swelling, and is covered with secretion. The *spleen* is usually enlarged in but slight degree, but by reason of an increase in its connective tissue it generally feels remarkably firm. The *kidneys* are increased in size, and usually present a bluish-red, almost blackish-red, color, which stands out sharply upon section, especially in the medullary portion. The condition is designated *cyanosis of the kidneys*, or, when the connective tissue has undergone hyperplasia

g-continued stasis and the kidneys have acquired a firmer
cyanotic induration of the kidneys. The *liver* also has in-
n size and it contains an excessive amount of blood. On
n consequence of marked dilatation and distention of the
eins with blood, a peculiar appearance is noted, suggest-
orm of an oak-leaf or a nutmeg, whence the name *cyanotic*
ver. If hyperplasia and contraction of connective tissue
e, the liver becomes harder and nodular upon its surface,
condition is then described as *cyanotic induration of the*
he *serosa of the stomach and intestine* is often marked by
active distention of the finer venous blood-vessels, and
mucous membrane swelling, brownish-red discoloration,
e secretion of mucus are striking. The *pancreas* also
enous distention, and frequently also unusual density in
ce of connective-tissue hyperplasia. Marked swelling
s hyperemia of the mucous membrane are usually pres-
urinary passages. The *sinuses of the dura mater* are, as
tended with blood. The *cerebrospinal fluid* is increased,
the surface of the brain and in the cortex the marked
of the veins is striking. Not rarely edema of the brain
dilatation of the cerebral ventricles have developed.

osis.—The prognosis in a case of weakness of the
m depends upon the **causes** and the **severity of the dis-**
covery can naturally be looked for only when the causes
able; but even then death may occur when the degree
ss of the cardiac muscle is so great that the heart is no
able of recuperating.

nent.—In all conditions of weakness of the myocar-
irst indication is the removal of the cause—**causal treat-**
addition the following **symptomatic treatment** should be
The patients should permanently occupy as nearly a
position in bed as possible, and confine themselves prin-
a *milk-diet.* Frequently all of the symptoms will dis-
r the institution of these measures alone. If marked
of the heart be present, an ice-bag should be placed
ly over the heart, and finally resort is had to *cardiac*
imulants. Among heart-tonics *digitalis-leaves* alone are
confidence, while their substitutes (tincture of stro-
caffein and sodium benzoate, caffein and sodium salicy-
s vernalis, convallaria majalis, spartein sulphate, and
quite unreliable agents. We prescribe digitalis-leaves
in the form of powder, and, if diuresis be deficient, in
n with diuretin (theobromin and sodium salicylate):

Powdered digitalis-leaves,	0.1 (1½ grains);
Diuretin,	1.0 (15 "). M.

e 10 such starch-capsules.
e: 1 every two hours.

In the use of all preparations of digitalis certain precautionary measures should be observed. At times *toxic manifestations* (vomiting, slowness of pulse, irregularity of pulse, diplopia, delirium) appear, which necessitate the withholding of the remedy. The *cumulative action* of digitalis must also be borne in mind. Some persons bear digitalis badly, and under such conditions *enemata of infusion of digitalis* have been employed. After too *protracted use of digitalis* the heart becomes accustomed to the remedy, and the activity of the drug is lessened. Patients taking digitalis should remain under the *constant observation of the physician*, in order that the remedy may be withdrawn at the proper time.

Stimulants may be prescribed with advantage when transient conditions of myocardial weakness are present, or when the myocardium has undergone such a degree of degeneration that digitalis is incapable of exerting its effect. A maintained influence is not to be expected under the latter conditions. Among agents of this class employed are especially camphor internally, or camphorated oil externally, ether, valerian-root, and ethereal tincture of valerian.

At times it is necessary to treat specially certain threatening symptoms; for instance, excessive cutaneous edema. Diaphoresis by means of pilocarpin hydrochlorate or sweat-chambers is usually not well borne, so that *multiple incisions of the skin* or *puncture* by means of Southey cannulas are preferably to be recommended.

DILATATION OF THE HEART.

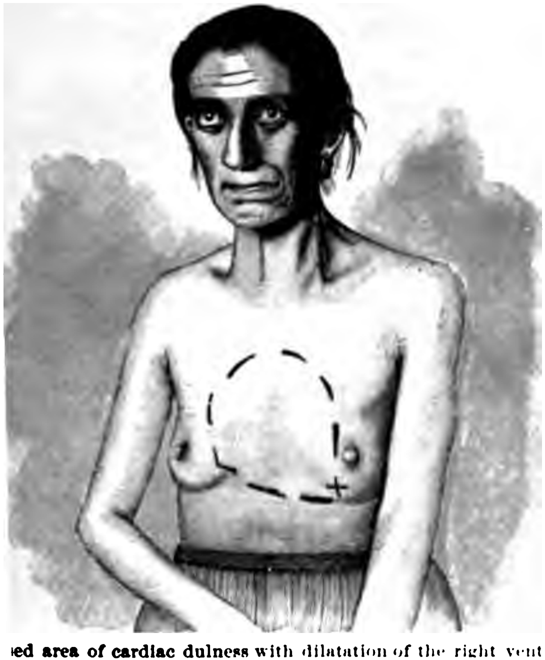
Etiology.—Dilatation of the heart may involve the right or the left side, or the entire organ. At times it is acute, at other times chronic in nature.

The conditions favoring dilatation of the heart are always provided when the *resistance* of the *myocardium is diminished* or when the *blood-pressure is increased in excessive degree.* Under the conditions first mentioned simple dilatation takes place, whereas under the second to the dilatation hypertrophy of the heart-muscle is, as a rule, superadded (so-called eccentric cardiac hypertrophy).

Diminished resistance of the heart-muscle and **cardiac dilatation** are observed in association with *infectious diseases, loss of blood and of bodily fluids, chlorosis, pernicious anemia, leukemia, pseudoleukemia*, and allied conditions. Frequently it involves only the right ventricle, which by reason of the greater thinness of its muscular wall appears of itself the more predisposed to dilatation. At times dilatation of the heart follows *poisoning* with mineral acids, alkalies, phosphorus, and arsenic. *Diseases of the myocardium* itself (cicatrices, fat heart) also give rise to dilatation of the heart. In some instances *congenital deficiency of resistance* on the part of the myocardium appears to be hereditary.

Dilatation (and hypertrophy) of the left ventricle in consequence of increased blood-pressure is observed especially in association with insufficiency of the aortic valve, stenosis of the aortic orifice,

icy of the mitral valve, aneurysm of the aorta, constric-
e aorta, arteriosclerosis, and contracted kidney. **Dilata-
hypertrophy) of the right ventricle** develops in association
fficiency of the mitral valve, mitral stenosis, insufficiency
lmonary valve, stenosis of the pulmonary orifice, insuffi-
the tricuspid valve, aneurysm and constriction of the
r artery, pulmonary emphysema, chronic bronchial
ronic pleurisy, kyphoscoliosis, and the like. At times
on of the one ventricle is gradually superadded a similar
ie other. The same condition arises especially in con-



ed area of cardiac dulness with dilatation of the right ventricle (**mitral
osis); from a photograph** (personal observation, Zurich clinic).

ith valvular disease of the heart when derangement of
on occurs.

ilatation of the heart in consequence of increased blood-
it times induced by bodily over-exertion ; for instance,
service, mountain-climbing, and excessive athletics.

on of the heart is most frequently encountered in *men
nced age*, which is readily comprehensible from a con-
f the etiology.

ms and Diagnosis.—Dilatation of the heart may
ecognized from the change in the area of cardiac dulness.
ition of the right ventricle the right border of the

(relatively) large area of cardiac percussion-dulness extends beyond the right sternal margin to the right (Fig. 2), while with dilatation of the left ventricle the left border of the heart extends beyond the left mammillary line to the left, in consequence of which the area of percussion-dulness at the same time acquires an oval outline (Fig. 3). In the presence of dilatation of the left ventricle the **apex-beat of the heart** attains unusual extent, being situated outside the left mammillary line and usually also lower than the fifth intercostal space on the left. When both ventricles participate in the dilatation the area of cardiac dulness exceeds its natural limits both to the left and to the right, and its outline becomes rather rectangular (Fig. 4).

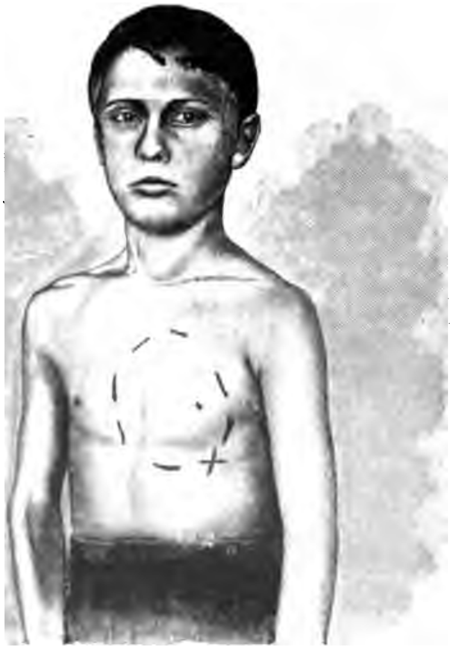


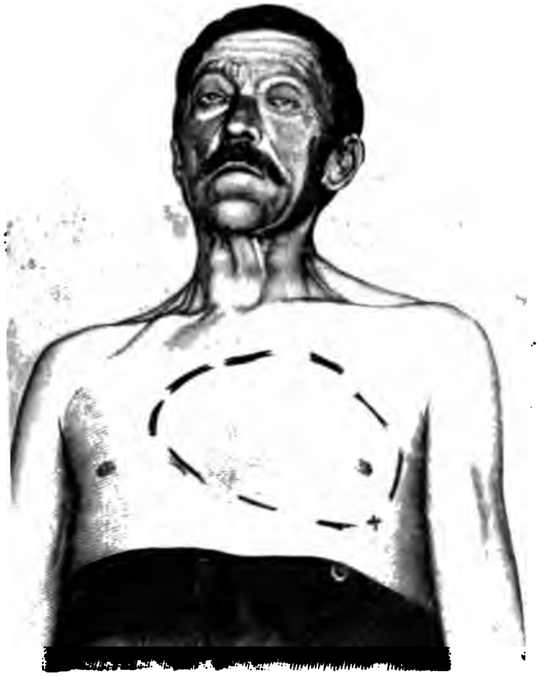
FIG. 3.—Increased area of cardiac dulness with dilatation of the left ventricle (insufficiency of the aortic valves; from a photograph (personal observation, Zurich clinic).

If in addition to dilatation of the heart *hypertrophy* also exists, this, if left-sided, will be attended with a heaving apex-beat, accentuation of the aortic second sound, and a hard radial pulse; while if right-sided, there will be accentuation of the pulmonary second sound, and frequently also unusually active systolic vibration over the sternum.

Individuals with dilatation of the heart may be entirely free from discomfort. This is especially so in cases in which, in addition to the dilatation, hypertrophy of the heart-muscle also exists, because both conditions tend to neutralize any possible

blood-pressure. If this do not occur, then the clinical
:ardiac **insufficiency** makes its appearance, with all of its
l dangers, and this takes place the more readily when
tion of the heart without hypertrophy is present.

mic Alterations.—Dilated chambers of the heart are
s for their **unusual size.** If the left ventricle is con-
lilated, the right at times forms only a sort of small
With dilatation of both ventricles the heart often
h considerable proportions that it has been designated



:ased area of cardiac dulness with dilatation of both sides of the heart
the mitral valves); from a photograph (personal observation, Zurich

bucardia (cor bovinum, enormitas cordis). In a
on the size of the heart should not exceed that of the
Changes in the shape of the heart also take place in
of dilatation, and if the left ventricle be involved
an oval shape, and if the right ventricle this assumes
r shape; if both sides are involved, the appearance
has been compared with a hunting-bag.

is.—The prognosis in case of dilatation of the heart
n the fact whether conditions of cardiac weakness
emoval is possible.

Treatment.—Treatment of dilatation of the heart is indicated only when manifestations of myocardial weakness appear, and is then to be conducted upon the lines laid down upon pages 23 and 24.

HYPERTROPHY OF THE HEART.

Anatomic Alterations.—Hypertrophy of the heart may, in the same way as dilatation, involve individual portions or the entire heart. As a rule, it is combined with dilatation of the heart—*eccentric hypertrophy of the heart.*

Concerning hypertrophy of the heart without alteration in the size of its cavities (*simple hypertrophy of the heart*), and even concerning such hypertrophy with diminution in size of the cavities (*concentric hypertrophy of the heart*), little of a certain nature is known. In many such instances the condition is probably one of contraction of an unaltered heart-muscle with the occurrence of death.

The hypertrophied segments of the heart-muscle are conspicuous for the **greater thickness of the wall,** and usually also for their **greater firmness.** At the same time the **weight of the heart** (in a healthy individual 300 grams) increases. Further, not only the walls of the heart, but also the papillary muscles, frequently take part in the hypertrophy, the latter becoming converted into thick, plump columns.

Histologically it can be shown that the hypertrophy of the heart-muscle is brought about partly through increase in the muscle-fibers (hyperplasia) and partly through increase in the size of individual muscle-fibers (pure hypertrophy). The intermuscular connective tissue may also be increased in amount.

Etiology.—Only **increase in blood-pressure** is with certainty known as a cause for hypertrophy (and dilatation) of the heart, and accordingly as this involves the aortic system or the distribution of the pulmonary artery, or both, will the left or the right ventricle or the entire heart be involved in the hypertrophy. In detail all of those conditions must be taken into consideration that have been mentioned on pages 24 and 25 as causes for dilatation of the heart.

Primary, idiopathic, or *essential hypertrophy of the heart* has also been variously spoken of. The designation is inappropriate, and should be replaced by *functional hypertrophy of the heart-muscle,* for idiopathic hypertrophy of the heart occurs in persons who, it is true, exhibit no anatomic alterations in their circulatory organs, but who have been compelled to perform unusually severe bodily work, or have made unusually large demands upon the heart-muscle through the ingestion of excessive amounts of fluid (Munich beer-heart). Such functional hypertrophy of the heart occurs especially in blacksmiths, locksmiths, seamen, wine-growers, mountain-climbers, and soldiers.

Whether *long-continued palpitation of the heart* and *excessive eating* give rise to hypertrophy appears not to have been demonstrated with certainty.

**l also to *toxic forms of cardiac hypertrophy*, resulting from exces-
coffee, tea, alcohol, tobacco, little that is reliable is known.**

toms and Diagnosis.—*Hypertrophy of the left ven-*
be recognized by the **heaving apex-beat,** the **accentuation**
ic second sound, and the **hard radial pulse,** conditions that
explained by the increased functional activity of the
le. If the thorax be sufficiently yielding, an unusually
ominence appears in the cardiac region, so-called **pre-
ging** (voussure) takes place.

ophy of the right ventricle is attended with accentuation
ionary second sound (valvular), for the greater the force
during the systole the blood is driven by the hyper-
entricle into the pulmonary artery, the greater will be
e with which it rebounds against the pulmonary valve
this to unfold with the succeeding diastole. Less regu-
is **unusually active vibration of the sternum,** beneath
right ventricle lies.

ophy of the heart is a desirable outcome in the pres-
culatory disturbances, and through its agency existing
an be overcome. There is great danger when the
ied heart fails in strength, for then there develops the
ture of *myocardial weakness,* which has already been
In the presence of *idiopathic,* or *functional, hypertrophy*
danger of cardiac weakness; and it is pathetic to wit-
sery of a robust, well-developed man, with an immense
ring from and eventually succumbing to the symptoms
weakness.

sis.—The prognosis in cases of hypertrophy of the
nds upon the *functional capability of the heart-muscle.*
rtrophied heart has a tendency to undergo connective-
fatty degeneration, and to lead up to increasing and
ardiac weakness. At times such conditions occur as
anifestations when extraordinary demands are made
yocardium in consequence of physical or mental excite-
olic and venereal excesses, and the like. Under these
he prognosis is more favorable, providing the over-
the heart has not exceeded a certain degree.

ent.—Treatment for hypertrophy of the heart is only
hen signs of *cardiac weakness* appear, and is then car-
he manner indicated upon pages 23 and 24.

FAT HEART (COR ADIPOSUM).

ic Alterations.—By fat heart in the clinical sense
l a morbid increase in the fatty tissue that is present
ht degree beneath the epicardium of a normal heart.

(relatively) large area of cardiac percussion-dulness extends beyond the right sternal margin to the right (Fig. 2), while with dilatation of the left ventricle the left border of the heart extends beyond the left mammillary line to the left, in consequence of which the area of percussion-dulness at the same time acquires an oval outline (Fig. 3). In the presence of dilatation of the left ventricle the **apex-beat of the heart** attains unusual extent, being situated outside the left mammillary line and usually also lower than the fifth intercostal space on the left. When both ventricles participate in the dilatation the area of cardiac dulness exceeds its natural limits both to the left and to the right, and its outline becomes rather rectangular (Fig. 4).

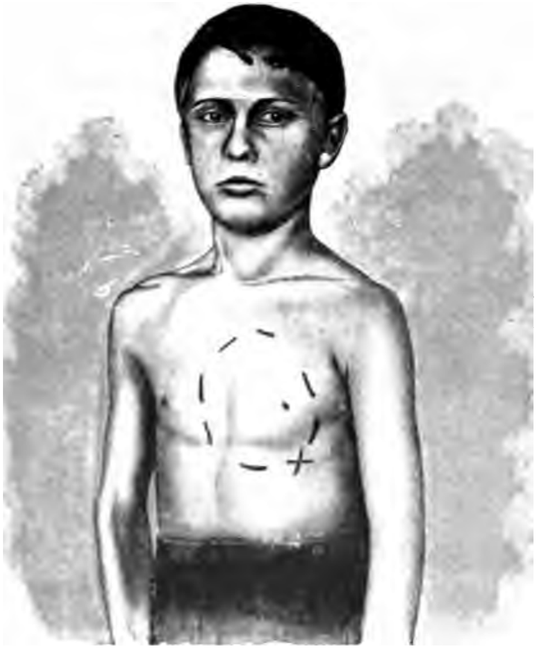
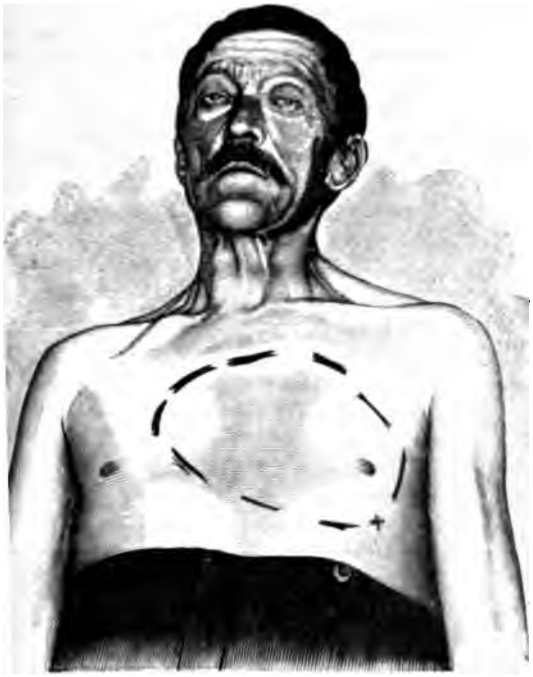


FIG. 3.—Increased area of cardiac dulness with dilatation of the left ventricle (insufficiency of the aortic valve); from a photograph (personal observation, Zurich clinic).

If in addition to dilatation of the heart *hypertrophy* also exists, this, if left-sided, will be attended with a heaving apex-beat, accentuation of the aortic second sound, and a hard radial pulse; while if right-sided, there will be accentuation of the pulmonary second sound, and frequently also unusually active systolic vibration over the sternum.

Individuals with dilatation of the heart may be entirely free from discomfort. This is especially so in cases in which, in addition to the dilatation, hypertrophy of the heart-muscle also exists, because both conditions tend to neutralize any possible blood-pressure. If this do not occur, then the clinical **cardiac insufficiency** makes its appearance, with all of its d dangers, and this takes place the more readily when tion of the heart without hypertrophy is present.

mic Alterations.—Dilated chambers of the heart are s for their **unusual size**. If the left ventricle is con- dilated, the right at times forms only a sort of small . With dilatation of both ventricles the heart often h considerable proportions that it has been designated



eased area of cardiac dulness with dilatation of both sides of the heart ' the mitral valves); from a photograph (personal observation, Zurich

, *bucardia* (cor bovinum, enormitas cordis). In a on the size of the heart should not exceed that of the **Changes in the shape of the heart** also take place in of dilatation, and if the left ventricle be involved an oval shape, and if the right ventricle this assumes r shape; if both sides are involved, the appearance has been compared with a hunting-bag.

is.—The prognosis in case of dilatation of the heart n the fact whether conditions of cardiac weakness removal is possible.

absorbed, and in its place there appears a **connective-tissue cardiac cicatrix.** The number and size of such indurations and cicatrices are very variable, and it is especially noteworthy that isolated, small foci of connective-tissue proliferation have given rise to the most distressing symptoms, while in other cases most extensive connective-tissue deposits have been unattended with symptoms. Often the heart-muscle undergoes hypertrophy.

The *detection of myocarditic alterations* requires the making of horizontal sections through the heart-muscle. Such changes are found with especial frequency in the *neighborhood of the apex of the heart.*

Etiology.—As *the later years of life* (beyond the age of forty) are frequently attended with endarteritic changes in the coronary arteries, it will be understood that the same period of life is also characterized by a tendency to the formation of connective-tissue cardiac cicatrices. At times, however, especially through the agency of certain **toxic influences,** presenile endarteritis and cicatrix-formation in the heart-muscle take place earlier in life, and excessive use of tea, coffee, alcohol, and tobacco, as well as gout, diabetes, contracted kidney, and chronic lead-poisoning, are not without reason mentioned as causes. Closely related to the toxic varieties of myocarditis are the **infectious varieties,** which have been observed in the sequence of syphilis, malaria, articular and muscular rheumatism. In some cases myocarditic cicatrices have developed in the sequence of **previous pericarditis** or **endocarditis** through extension of the inflammatory process to the heart-muscle. **Injuries of the precordium** or **of the heart-muscle** itself are less common causes of myocarditic cicatrix-formation. With regard to the **influence** of **exposure to cold** nothing definite is known. That myocarditis is most common in *men of advanced years* should not excite surprise in view of the etiology.

Symptoms.—Myocarditic cicatrices give rise to symptoms only when they interfere with the **action** and the **functional capability of the heart-muscle.** Palpitation, irregularity of the action of the heart, and a sense of oppression and of fear are usually among the earliest symptoms, and are followed by manifestations of progressively increasing and gradually insurmountable cardiac weakness and general stasis. The condition may persist for many months and years, and hence has also been designated *chronic myocarditis. Overwhelming stasis* is the most common cause of death. At times *death takes place suddenly* in consequence of a variety of causative factors; sometimes it may result from over-distention of the heart and *cardiac paralysis.* In the presence of marked narrowing of the coronary arteries in consequence of endarteritis, *sudden occlusion* by thrombi or, as it appears, also at times by abnormal angulation of the vessel, will under some conditions cause sudden death in consequence of *anemia of the heart-muscle.* Much less

a sudden fatal termination occurs from *rupture of the*
rebral embolism, the latter originating from thrombi in

aneurysm of the heart may be mentioned as on the whole an
sequel of chronic myocarditis. It results in consequence of
atation of a connective-tissue cicatrix, and may exceed the size
t. The condition is scarcely susceptible of positive diagnosis.
cardiac dulness of *unusual extent and irregular shape*, *anomalous*
n the precordium, and *murmurs* in the same situation are the
ut not unequivocal, symptoms. Death may result from *rupture*
rysm, or from *embolism* arising from thrombi in the aneurysm.

osis.—Chronic myocarditis can never be diagnosed
nty. Only existing disturbances of cardiac activity and
be recognized with certainty; but that these are due to
mation in the heart-muscle can under the most favor-
ions be correctly interpreted only when there is reason
that all other causes for the manifestations named can
l.

osis.—The prognosis in cases of chronic myocarditis is
e, for no remedy is capable of causing disappearance of
es. On the contrary, there is danger of the formation
atrices, with steadily increasing cardiac weakness.

ent.—**Prophylactic treatment** is applicable to the toxic
myocarditis, and should be directed to the correction
philis, lead-poisoning, and the use of alcohol, tobacco,
tea. **Causative treatment** is indicated when arterio-
syphilis is present. Under both of these conditions
iodid should be prescribed (5.0 to 200—75 grains to
ces; a tablespoonful thrice daily). Naturally, prophy-
causative treatment overlap, as a rule, without sharp
Symptomatic treatment aims principally at the cor-
xisting derangement of cardiac action, and of diminu-
rt-strength, and can be carried out in accordance with
ons made on page 23 and 24.

OF THE MYOCARDIUM (NEW-GROWTHS OF THE HEART).

mations in the heart-muscle (carcinoma, sarcoma, myxoma,
a, myoma) occur rarely, and can never be diagnosed with
all tumors are *unattended with symptoms*, while *larger*, by dis-
the heart-muscle, give rise to *disturbed action of the heart*
se of the heart-muscle or to emboli, when fragments of the
re broken off and are carried with the blood-current into
ries. At times *symptoms of a valvular lesion* appear from the
the tumor toward a valve, whose activity is thus interfered
such conditions a probable diagnosis may be possible when
nonstrable in peripheral organs. If the tumor of the myo-
given rise to an increase in the area of cardiac dulness, there
nfusion with *pericarditis*.

Treatment is futile, and should be confined to the relief of individual symptoms. The prognosis is therefore invariably unfavorable.

ECHINOCOCCUS OF THE HEART.

Echinococcus of the myocardium is an extremely rare affection, which experience has shown to involve the right ventricle most frequently. At times the cysts rupture, and accordingly as the discharge takes place into the right or the left ventricle they give rise to *emboli* in the distribution of the pulmonary artery or the aorta. Sudden occlusion of an orifice of the heart may lead to sudden death.

Treatment is hopeless.

DEXTROCARDIA.

In some persons the heart is not situated on the left but on the right side of the thorax, so that the cardiac apex likewise is directed toward the right axillary region and the arch of the aorta winds around the right bronchus. The condition is an *indifferent congenital* one, and it may be present in the most healthy and most robust individual. The remaining organs may also participate in the displacement, so that the left lung has three lobes, the liver is situated beneath the left half of the diaphragm, the spleen beneath the right, the cardia of the stomach to the right of the vertebral column, the pylorus to the left, etc. Such a condition is known as *situs viscerum perversus s. inversus.* Care must be taken not to confound dextrocardia with *displacement of the heart* in consequence of *left-sided pleurisy with effusion.* It may also happen that after absorption of a left-sided pleural effusion has taken place the heart is bound in its abnormal situation by adhesions; but under such conditions the left side of the chest becomes retracted. Marked contraction of the right lung also may give rise to displacement of the heart in the right half of the chest; but then the right side of the thorax becomes retracted.

CARDIORHEXIS (RUPTURE OF THE HEART).

Etiology.—Rupture of the heart, apart from *injury of the heart*, probably occurs only when the integrity of the heart-muscle has been impaired by previous disease. Among such causative conditions are especially *fat heart, myocarditis, tumor-formation and echinococcus of the heart, aneurysm and abscess of the heart-muscle*, and *softening resulting from occlusion of the branches of the coronary arteries.* At times aneurysm of a coronary artery leads to attenuation and rupture of the myocardium. As may be understood, rupture will readily take place when the blood-pressure has undergone increase. In accordance with this fact, rupture of the heart has not rarely been observed in connection with *congenital narrowing of the isthmus of the aorta*, and the condition has arisen frequently after psychic and physical exertion. The causes named explain why rupture of the heart is most common in men of advanced years.

Anatomic Alterations.—When rupture of the heart occurs blood is usually poured out in large amount into the pericardial sac, so that this presents the appearance of an intensely distended bladder of a bluish-black color. The site of laceration is most frequent in the left ventricle, close to the apex of the heart, and the opening as a rule is occluded by blood-clots. The tear usually takes place in the longitudinal axis of the fibers of the heart-muscle, frequently exhibits a sinuous course so that the epicardial and endocardial openings are not in apposition, and at times presents ramifications in its course.

Symptoms and Diagnosis.—Rupture of the heart may cause *sudden death*. At other times the victim cries out that something has ruptured in the precordium; he is seized with an indescribable fear and a premonition of death, presents pale and distorted features, and exhibits the *signs of internal hemorrhage*, with *rapidly increasing enlargement of the area of cardiac dulness* in consequence of the effusion of blood into the pericardial cavity. Under such conditions also death usually takes place within a short time, partly in consequence of the hemorrhage, partly in consequence of interference with the action of the heart, at times also as the result of shock.

Prognosis and Treatment.—The prognosis is unfavorable, for scarcely any good can be hoped for from the application of *an ice-bag to the precordium* and the administration of *stimulants* and *styptics*.

II. DISEASES OF THE ENDOCARDIUM.

ACQUIRED VALVULAR DISEASE OF THE HEART.

Etiology.—Acquired valvular disease of the heart is one of the *most common diseases of the heart* and diseases in general. At times the condition is one of *defect in closure or insufficiency of a valve*, at other times one of *constriction or stenosis of a valvular orifice*. Often, however, both conditions are present together in one and the same valvular apparatus, and possibly only one may preponderate. If several valvular apparatus are simultaneously diseased, the condition is designated *combined valvular disease of the heart*.

In the majority of cases valvular disease of the heart is dependent upon anatomic alterations. Much less commonly the valvular apparatus is itself healthy, but is insufficient to effect closure on account of morbid dilatation or distention of the valvular orifice. Such a condition is designated *relative valvular insufficiency*.

Acquired valvular disease of the heart affects almost exclusively the *valves of the left side*, especially the mitral, which most frequently is the seat of imperfect closure. Right-sided valvular disease of the heart is, as a rule, congenital. An exception is constituted by relative tricuspid insufficiency, which develops not rarely in the sequence of mitral disease, and with especial frequency after mitral stenosis.

The most frequent cause of acquired valvular disease is **endocarditis**. This usually develops after **infectious diseases**, and among these acute articular rheumatism is most prominent. *Acute articular rheumatism is thus the most common cause of acquired valvular disease of the heart.* In childhood scarlet fever is an important cause.

At times valvular disease of the heart does not develop till late in life, when the aorta and the aortic valves, less commonly the mitral valve, are attacked by **arteriosclerosis**, which leads to thickening, calcification, and contraction of the valvular tissue. These alterations are frequently looked upon as constituting *chronic endocarditis*.

Only rarely are valvular lesions of the heart induced by **aneurysms of valves, neoplasms upon the valves** or in their proximity, or by **laceration of valves.** Laceration may occur also in healthy valves in consequence of excessive physical exertion, and has been observed in all the valves of the heart, with the exception of the pulmonary.

Excessive distention of the heart is considered the cause for relative valvular insufficiency. As has been mentioned, relative tricuspid insufficiency not rarely develops in association with mitral disease. In individuals engaged in heavy physical exertion over-distention of the left ventricle develops at times, with mitral insufficiency, and which may recede with care.

Acquired valvular disease of the heart occurs more frequently in *adults* than in children, because articular rheumatism occurs but rarely in the latter. Clinical statistics are almost unanimous in indicating that valvular heart-disease is more common in *women* than in men. In certain *regions* at the seaside, and in damp and foggy places, valvular lesions of the heart are encountered with especial frequency.

Anatomic Alterations.—The anatomic alterations attending acquired valvular heart-disease consist mainly in deposits, thickenings, even calcification, contraction, adhesions, or detachment of valvular tissue. Anatomic alterations are wanting only in cases of relative valvular insufficiency. In addition to the endocardium, the **heart-muscle** itself is almost always found dilated and hypertrophied in certain segments. The situation of the parts thus affected depends upon the nature of the valvular lesion. As most patients with valvular heart-disease die in consequence of weakness of the heart-muscle, the internal viscera, as a rule, exhibit the appearances of **venous stasis**, such as have been described on page 18.

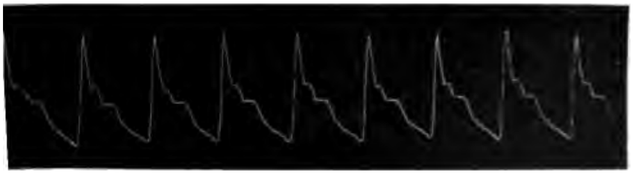
It is by no means always easy to decide at autopsy as to the nature of a valvular lesion of the heart. To demonstrate the existence of insufficiency of the aortic or pulmonary valve a strong stream of water is permitted to fall from a considerable height into the trunk of the arteries named. If the valve be incompetent, the water quickly flows into the ventricle, while if it be competent the water will be supported. In the presence of narrowing of the aortic or pulmonary orifice septum-like adhesions of the valves with one another or cicatrix-like contractions of the orifice are frequently encountered. In deciding as to the existence of incompetency or stenosis of the mitral or tricuspid valve or orifice the knowledge that in healthy adults the auriculoventricular orifices are permeable by the middle and index fingers will serve as a guide.

toms.—For the recognition of acquired valvular disease
rt the *local cardiac alterations* furnish the indication.
general disturbances of the circulation have also devel-
these are most frequently manifested as *general venous*
much less commonly by *emboli.* We shall consider these
ps of symptoms successively.

**ardiac Manifestations associated with Acquired Valvular
the Heart.**—1. *Aortic Insufficiency; Aortic Regurgita-*
principal symptoms of aortic valvular insufficiency
an *aortic diastolic murmur,* in *dilatation* and *hyper-*
he left ventricle, and in an *accelerated, full, hard radial*

rtic valves are incompetent, a portion of the aortic blood, during
of the left ventricle, flows back into this chamber. As this
nparison with the lumen of the aorta, represents a sudden dila-
blood-stream, there result eddies or whirls, which give rise
o a *diastolic murmur.* This is frequently louder over the middle
m than over the area of auscultation for the aorta (right second
pace), because the left ventricle is in closer proximity to the
sternum. The murmur may also be transmitted with dimin-
ity to the other valvular areas, to the carotid, and for consider-
upon the thorax. Only rarely is it appreciable at some distance
ent, and more frequently it is palpable as a *diastolic purring*
sement cataire).

that if the left ventricle shall receive the blood regurgitated
in addition to that sent to it from the auricle, *dilatation of the*
take place, and this adjusts itself to the degree of insuffi-
is recognized by the appearance of the apex-beat outside the
ry line and below the left fifth intercostal space, and the presence
d area of cardiac dulness extending beyond the left mammil-
26, Fig. 3). As, however, the heart-muscle will be compelled
stole to send more blood into the aorta than under normal con-
hus to perform a greater amount of work, *hypertrophy of the*
ill be a further necessary sequel. This is recognized by the
beat. The radial pulse exhibits the qualities of a large, hard,
lse, because all of the arteries are filled with an abnormally
of blood under unusually high pressure, and during the dias-
rt the blood flowing back against the semilunar valves is not



graphic tracing from the radial artery in a case of aortic valvular insuffi-
ciency (personal observation, Zurich clinic).

into the arteries, but is taken up by the left ventricle. A
c tracing exhibits the peculiarities of the pulse in a distinc-
ig. 5).

to the manifestations described, secondary conditions are
e in cases of aortic valvular insufficiency, which, while they

complete the clinical picture, are of subordinate importance with regard to the diagnosis. Among these may be mentioned: 1. *Precordial bulging and abnormally active and widely extended cardiac pulsation.* These result from the left-sided hypertrophy of the heart. 2. *A systolic heart-murmur*, possibly the result of irregular contractions of the myocardium. This may be referred to simultaneous stenosis of the aortic orifice only when the pulse is not rapid but retarded. 3. *Heaving and vibration of the carotids and a carotid systolic murmur.* Excessive distention of the arteries readily leads to irregular vibrations of the vessel-wall. The remaining secondary phenomena are also attributable to excessive distention of the arteries with blood—such as: 4. *Arterial murmurs;* short cardiac-systolic murmurs are heard also in the smaller arteries; for instance, the radial or the palmar arch. 5. *Visible pulsation* in the smaller arteries; for instance, the temporal and the retinal. 6. *Visible and palpable pulsations in various organs* (uvula, soft palate, penis, and enlarged spleen). 7. *Visible capillary pulsation* (to be looked for in the finger-nails or at the margin of an area of skin reddened by rubbing). 8. *Traube's double murmur in the femoral artery.* On careful application of the stethoscope a systolic and a diastolic murmur are heard over the femoral artery. 9. *Duroziez's pressure-sign in the femoral artery.* On application of a certain amount of pressure with the stethoscope a systolic and a diastolic murmur can be heard over the femoral artery.

Persons with aortic valvular insufficiency bear their disease at times for years without noteworthy disturbance, because the left ventricle with its hypertrophied musculature is capable of neutralizing the disturbances in the circulation as completely as possible. Annoying palpitation of the heart is frequently the first symptom. Not rarely *repeated epistaxis* takes place. To this occurrence the *pallid appearance* of the patients has been attributed, but this is also observed in those who have never suffered from nose-bleed. A *tendency to cerebral hemorrhage* also is assigned to individuals with aortic valvular insufficiency.

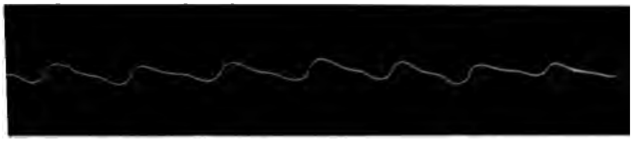
2. *Aortic Obstruction; Aortic Stenosis.*—In the presence of constriction of the aortic orifice there first occurs a *systolic murmur* as soon as the blood, during the systole of the left ventricle, has passed the narrowed aortic opening and has entered the wide first part of the aorta, as sudden enlargement of the current-bed leads to the formation of blood-whirls. The murmur is often marked by especial intensity, so that it is transmitted from the right second intercostal space, the point of auscultation for the aorta, to the remaining valves and still farther, and it is not rarely palpable as a *systolic purring tremor.*

An obstruction to the blood-stream at the beginning of the aorta must necessarily lead to *hypertrophy and dilatation of the left ventricle*, in order that the obstruction may be overcome. The dilatation will, however, be but slight in comparison with that which develops in association with aortic valvular insufficiency, because regurgitation of blood into the ventricle does not take place. The condition of the radial pulse is important, exhibiting on palpation and in a sphygmographic tracing the characters of an infrequent, small, and retarded pulse (page 39, Fig. 6).

Smallness and retardation of the pulse may be explained by the slow entrance of the blood into the aorta, while the slow pulse may be referred to insufficient distention of the coronary arteries of the heart.

with aortic stenosis usually exhibit *a pallid appearance*
ence of imperfect filling of the arterial system with
they not rarely suffer from *attacks of syncope* when
iemia is excessive.

l Insufficiency; Mitral Regurgitation.—Incompetency
ral valve gives rise to a *systolic murmur*, which is usu-



ographic tracing of a radial pulse from a case of stenosis of the aortic
orifice (personal observation, Zurich clinic).

at the apex of the heart, less commonly over the
orifice, and to *dilatation and hypertrophy of both*

mitral valve is incompetent the blood during the systole of
cle is propelled not only into the aorta, but in part back again
uricle. In consequence, the left auricle is compelled to receive
arge amount of blood; namely, that from the pulmonary veins
rgitated from the ventricle. This is possible only if *dilatation*
icle takes place, although this first physical consequence of
r insufficiency is without clinical significance. As the left
parison with the contracting left ventricle, represents a sud-
of the blood-stream, and, besides, as two currents of blood
o the auricle at the same time, blood-whirls must be gener-
re appreciable acoustically as a *systolic murmur*, and if of
sity are also palpable as a *systolic purring tremor*. The blood
e pulmonary veins into the left auricle finds this cavity par-
h blood regurgitated from the left ventricle, and stasis must
. This blood-stasis extends from the area of the pulmonary
f the pulmonary capillaries and the pulmonary artery, and
ight ventricle. In order that the latter may overcome the
and hypertrophy of the right ventricle must take place. This
nifested by *enlargement of the area of cardiac dulness beyond*
margin, while the hypertrophy leads to *accentuation of the*
d sound, and not rarely also to *unusually active vibration of*

ence of mitral valvular insufficiency increased functional
de not only upon the right, but also upon the left ventricle,
ystole a greater amount of blood (normal amount of auric-
the regurgitated blood) must be sent not only into the tube
but into two cavities (aorta and left auricle). In order to
e greater amount of blood *dilatation of the left ventricle* will
order to propel the blood *hypertrophy of the left ventricle* will
In accordance with the dilatation of the ventricles the
dulness becomes enlarged both to the right and to the left
). The apex-beat of the heart is then generally extensive,
outward beyond the left mammillary line and often also
heaving, the last in consequence of hypertrophy of the left

4. *Mitral Obstruction; Mitral Stenosis.*—A *presystolic murmur* and *dilatation and hypertrophy of the right ventricle* are the principal local symptoms of mitral stenosis.

The *presystolic murmur* is generated by the formation of whirls in the blood passing during the diastole of the left ventricle from the left auricle through the constricted mitral orifice into the large left ventricle. Frequently a *presystolic purring tremor* is also palpable.

Not rarely the *presystolic murmur appears loudest at the beginning and at the close of the diastole,* for at the beginning the left ventricle is still empty, and the formation of the blood-whirls therefore very active; and toward the end of the diastole the left auricle undergoes contraction, and thus gives rise to renewed activity in the formation of blood-whirls.

Dilatation and hypertrophy of the right ventricle are a natural consequence of the obstruction that the blood of the left auricle encounters at the constricted mitral orifice and of the resulting stasis, which extends through the pulmonary veins, the pulmonary capillaries, and the pulmonary arteries to the right ventricle.

At times the *diastolic sound over the aorta and the pulmonary artery* appears duplicated. This is explained by the fact that the pressure in the aorta and the pulmonary artery has undergone such pronounced alteration in consequence of the mitral stenosis that the semilunar valves of the aorta close earlier than those of the pulmonary artery.

The *systolic sound at the apex of the heart* frequently is unusually clear and loud, perhaps in consequence of the fact that the difference in tension of the mitral valvular leaflets during the systole and diastole of the left ventricle in cases of mitral stenosis is unusually great.

5. *Tricuspid Insufficiency; Tricuspid Regurgitation.*—In addition to a *systolic murmur over the tricuspid valve* and *dilatation and hypertrophy of the right ventricle,* a *positive venous pulse* especially is indicative of incompetency of the tricuspid valve, and from the presence of which the valvular lesion can often be at once suspected.

In cases of incompetency of the tricuspid valve a portion of the ventricular blood will flow back into the right auricle during the systole of the right ventricle. As, however, the right auricle must also receive the blood from both venæ cavæ, it must undergo increase in capacity. *Dilatation of the right auricle,* therefore, takes place, but this plays clinically only a subordinate *rôle.* As soon as the blood, during the systole, is thrown back from the right ventricle into the right auricle blood-whirls ensue in the latter, and are appreciable acoustically as a *systolic murmur,* for the still empty auricle, as compared with the contracting right ventricle, represents a sudden expansion of the blood-stream, and, in addition, it is distended by two currents of blood (regurgitated blood from the ventricle and blood from the venæ cavæ). *Dilatation of the right ventricle* will, therefore, develop, because the right ventricle, during the systole, must receive, in addition to the normal auricular blood, also that forced back into the right auricle during the preceding systole. As, however, the right ventricle, during the systole, must again propel this large amount of blood, and must pump into two orifices of exit (pulmonary artery and right ventricle), it must perform a greater amount of work, and accordingly *hypertrophy of the right ventricle* takes place.

The blood forced back from the right ventricle into the right auricle during the systole, presupposing sufficient power on the part of the heart-muscle, is driven into the superior and inferior venæ cavæ, and thus gives rise to a *positive venous pulse.* In the distribution of the superior vena cava

enous pulse first appears in the neck, and earliest in the external
1. The blood surging upward encounters resistance within the
gular vein at the point where the semilunar valves of the bulb
are situated, and in consequence, between the inferior points of
e sternomastoid muscle just above the sternoclavicular articu-
ythmic cardiac-systolic vibration is visible, the *bulbar pulse.*
o be felt as a short blow; and on auscultation a short sound can
e *bulbar valvular sound.* Soon, however, the orifice of the bulb
greatly distended that its valves are no longer capable of effecting
relative insufficiency of the bulbar valve takes place. The blood
from the right ventricle into the superior vena cava can now be
he internal jugular vein itself, and there results a venous pulse
l. Above the bulb of the jugular vein a cardiac-systolic bulbar
hen not rarely audible, and at times a systolic *bulbar thrill* is
e. If, however, the jugular vein is greatly distended by the
ing blood-stream, a cardiac-systolic *venous murmur* results.

e venous pulse may also be visible in the smaller branches of
vena cava, especially in the *large veins of the arm and the face.*
ic venous pulse results when the blood from the right ventricle
the right auricle and the inferior vena cava with every systole,
cally fills the distribution of the hepatic veins with blood.
n can be seen and still more frequently felt with the hand
nds are placed upon the anterior and posterior aspects or upon
left sides of the liver.

cases the blood-wave finds its way into the femoral artery.
s rise (just below Poupart's ligament), in the same manner as
l jugular vein, to a *femoral bulbar pulse*, a *venous valvular mur-*
alvular insufficiency, a *femoral venous pulse*, and even a *saphenous*
r murmurs and a *femoral venous murmur.*

enous pulse appears only *temporarily*, disappearing when the
heart has grown so slight that regurgitation of a visible and
e-wave does not occur; and it will not appear in the internal
nd the femoral vein as long as the bulbar valves close, or with
tasis become again competent.

ring condition may be mistaken for the cervical venous pulse:
movements in the veins of the neck. These cease when respira-
nded; 2. *Pulsatile movements imparted by the carotid artery.*
the artery interrupts the pulsation in the carotid and at the
so that in the veins of the neck; 3. *Negative venous pulse.*
stole of the right auricle, and the diastole of the right ven-
od stagnates in the superior vena cava and gives rise to a
ounced distention of the cervical veins; but in the first place
does not occur, like that of the positive venous pulse, at the
stole, but during the diastole of the heart, so that a negative
arotid pulse are not synchronous but alternating; and besides,
el is compressed the negative venous pulse necessarily dis-
the point of compression, while the positive venous pulse
l by this manipulation, and frequently becomes even more

nous pulse might be confounded with pulsation imparted to
the abdominal aorta, but under such circumstances there
simple elevation and depression of the organ, while in the
ositive hepatic venous pulse a *general* cardiac-systolic enlarge-
er takes place.

id Obstruction; Tricuspid Stenosis.—Tricuspid steno-
mmonly rare valvular lesion of the heart which gives
***ystolic murmur** over the tricuspid orifice* and to *dilata-*

tion and hypertrophy of the right auricle. The latter leads to increase in the area of cardiac dulness toward the right.

7. *Pulmonary Insufficiency; Pulmonary Regurgitation.*—Insufficiency of the pulmonary valve is recognized by a *diastolic murmur over the pulmonary orifice* (left second intercostal space close to the left sternal border) and by *dilatation and hypertrophy of the right ventricle.* The development of these alterations is exactly the same as that of the corresponding manifestations attending insufficiency of the aortic valve, but referable to the right instead of the left heart. This form of valvular lesion also is rarely observed.

8. *Pulmonary Obstruction; Pulmonary Stenosis.*—Constriction of the pulmonary orifice is *usually a congenital and but seldom an acquired valvular lesion of the heart.* A *systolic murmur over the pulmonary orifice,* frequently a *systolic purring tremor,* and *dilatation and hypertrophy of the right ventricle* are the distinctive manifestations. These arise in the same manner as the corresponding alterations associated with aortic stenosis, but with reference to the circulation through the pulmonary artery and the right ventricle.

General Venous Stasis attending Valvular Lesions of the Heart, Weakness of the Heart-muscle, or Derangement of Compensation.—The circulatory disturbances to which a valvular lesion of the heart gives rise can only be neutralized to a certain degree by dilatation and hypertrophy of certain portions of the heart, which thus *compensate for the valvular lesion.* As soon as the vigor of the heart-muscle becomes impaired the condition arises that is designated *derangement of compensation*, and there develops the clinical picture of general venous stasis, as described on page 18. Such a derangement of compensation may be transitory and the result of *disturbance of innervation* or of *over-distention of the heart.* It occurs also in the sequence of mental and physical exertion, of alcoholic and venereal excess, injudicious use of tea, coffee, tobacco, and the like. Many persons suffer repeatedly from attacks of derangement of compensation, which again subside under proper precautions. Nevertheless, in spite of all care, derangement of compensation will become more and more marked, because an hypertrophied heart-muscle manifests a *tendency to fatty and connective-tissue degeneration,* with increasing cardiac weakness. Such conditions are usually incurable and progressive, and eventually cause death.

Embolic Alterations attending Valvular Lesions of the Heart.—In addition to derangements of compensation, patients suffering from valvular lesions of the heart are especially exposed to danger from *emboli.* These result from the detachment of deposits upon the valves, which enter the blood-current and are with it carried to the arteries of remote organs, in the smaller branches of which they finally lodge as emboli. Emboli occur most frequently in

s of the spleen, the kidney, the extremities, and the
e they are much less common in the hepatic arteries,
eric arteries, and the retinal artery. The sudden onset
iptoms and the usually sudden pain are among the dis-
itures of embolism.

i *of the splenic artery* is characterized by the appearance of
in the splenic region, acute enlargement of the spleen, tender-
organ on pressure, and at times by audible and palpable peri-
tion-murmurs. With the occurrence of the embolism vomiting,
ver may be present. *Embolism of the renal artery* may likewise
with vomiting, chill, fever, and sudden pain in the renal region.
the urine contains blood—hematuria. *Embolism of the hepatic*
' rare, and gives rise to marked jaundice and rapid diminution
' the liver, thus to the clinical picture of acute yellow atrophy
Embolism of the mesenteric artery causes sudden pain in the
ritonitis, and bloody stools. *Embolism of the artery of an ex-*
ended with loss of pulsation and with coldness of the affected
w the point of occlusion, and if an adequate collateral circula-
e established gangrene sets in. *Embolism of the cerebral arteries*
ion in the distribution of the left carotid, which represents the
of the aorta, and thus leads to occlusion of the left Sylvian
patients are usually rendered unconscious with the occurrence
ism, and suffer a stroke or attack of apoplexy. When con-
urns right-sided palsy will be observed, hemiplegia dextra, and
o aphasia. *Embolism of the retinal artery* causes sudden blind-
imination with the ophthalmoscope the macula lutea presents
e of a cherry-red spot in the anemic retina.

sis.—The diagnosis of a valvular lesion of the heart
only when endocardial murmurs are demonstrable on
At times the blood-pressure is so low as not to gen-
blood-whirls, and when a valvular lesion is suspected
nay be made to increase artificially the blood-pressure
king or by movement of the body to and fro. It is
e to auscultate the heart in both the upright and the
osition of the body, at the end of inspiration and of
nd to note the presence of possible murmurs.
istolic murmurs scarcely occur except in association
r lesions, systolic murmurs may also be present in
ith *febrile* and *anemic* states as *accidental murmurs.*
of valvular lesion of the heart may therefore be made
sence of such murmurs only when some portions of
be shown to be dilated and hypertrophied. Dilata-
not sufficient, as it may be the result also of the fever
. Besides, it should be borne in mind that accidental
ost never give rise to purring tremor.

an additional important adjunct in differential diagnosis,
e, is not present in association with valvular lesions, is the
lse is least frequent in the recumbent posture, more frequent
sture, and most frequent in the standing posture.

orrect localization of heart-murmurs the rule should be rigidly observed that murmurs are generated at those valves over which they are heard with greatest intensity. As has been mentioned, exceptions occur at times in the case of insufficiency of the aortic and the mitral valve. If there be doubt whether a murmur is aortic or pulmonary, it should be noted whether it persists as the stethoscope is moved toward the right or the left. In the first instance it is generated in the aorta. If, however, it disappears as the stethoscope is moved toward the right, while it persists with the movement of the instrument toward the left, it is pulmonary in origin. The same principles apply to the differentiation between mitral and tricuspid murmurs.

Combined valvular lesions of the heart may be readily recognized when the one lesion gives rise to a systolic and the other to a diastolic murmur. If, however, murmurs occur at the same time, these may be transmitted from one valve to another, and the differentiation would depend upon the fact whether the murmurs exhibited different acoustic characters at different situations, and whether the dilatation and the hypertrophic alterations in the heart-muscle indicated a combined valvular lesion. In order to diagnose an associated aortic stenosis together with the existence of aortic insufficiency from the presence of a systolic murmur, the fact should be taken into consideration, as has been mentioned, whether the radial pulse is retarded or not. The combination of a valvular lesion with tricuspid insufficiency is indicated by a positive venous pulse.

The **anatomic alterations** are, as a rule, not susceptible of diagnosis. As the clinical diagnosis relates only to the functional disturbance of the diseased valve, it not rarely occurs that at the autopsy the impression is created of an existing stenosis rather than of the diagnosed insufficiency, and *vice versa*.

Prognosis.—Valvular lesions of the heart are insusceptible of cure by the administration of drugs, and the prognosis is correspondingly unfavorable. In rare instances **spontaneous recovery** takes place, perhaps because of the gradual disappearance of deposits upon valves, or the stretching of healthy valves, or the retraction of dilated orifices. With the necessary rest of body and mind, and an *appropriate mode of life*, valvular lesions of the heart may be survived for decades. All *febrile diseases* and in women *pregnancy* readily induce cardiac weakness and derangement of compensation. Valvular lesions of the heart acquired in childhood are attended with the danger of derangement of compensation at the period of puberty. Sufferers from valvular heart-disease are but rarely attacked by *pulmonary tuberculosis*.[1] An exception is con-

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[1] This statement would seem to require modification. While pulmonary tuberculosis may be less common in association with valvular disease of the heart than with other conditions, it can scarcely be said to be rare. The subject would appear to be worthy of further investigation.—A. A. E.

pulmonary stenosis, in connection with which pulmonary
s occurs frequently, probably in consequence of im-
rition and diminished resistance on the part of the

ent.—There is *no remedy for valvular disease of the*
late **saline baths** have been much employed, especially
auheim; but recovery, even under these conditions,
exceptionally. The greatest importance in the treat-
atient suffering from valvular disease should be placed
t physical and psychic mode of life, and upon a **light and**
us diet, especially a judicious milk-diet. Medicaments
rescribed only when threatening symptoms arise. Most
weakness of the heart-muscle and derangements of
n must be corrected, in accordance with the rules laid
page 23 and 24. **Digitalis** is the most trustworthy agent.

CONGENITAL HEART-DISEASE.

ic Alterations.—Congenital lesions of the heart are
f cardiac disease. The condition most commonly found
stenosis of the pulmonary orifice. Lesions of the tri-
especially those of the valves of the left side of the
nusually rare occurrences. Congenital heart-lesions,
t only involve the valves and orifices of the heart, but
give rise to **patency of the foramen ovale,** to **deficiency**
uricular and the interventricular septum, to **patency,**
neurysmal dilatation of the duct of Botal, or to **transpo-**
great vessels, the aorta arising from the right and the
rtery from the left ventricle. Frequently *several con-*
s of the heart exist in association; especially with pul-
sis the foramen ovale and the duct of Botal not rarely
ous, so that the blood may pass from the right into the
nd from the aorta through the duct of Botal into the
rtery. Naturally, such persistence of fetal circulatory
cessary even when the pulmonary orifice is not merely
ut entirely closed.

y.—Nothing definite is known as to the causes of con-
s of the heart. Among some that have been men-
chic disturbances and injuries during pregnancy, articular
luring pregnancy, syphilis, and **consanguinity** in the
times several children in the same family have been
ngenital lesions of the heart. Experience has shown
fer more commonly than girls. Most frequently the
ears to be a **defect in the development of the heart,**
organ, by reason of the numerous changes in shape
h it passes during fetal life, appears to be particularly
times other developmental defects have been observed

in association with congenital lesions of the heart; for instance, harelip, club-foot, congenital deaf-mutism. In other instances the condition may be the consequence of **fetal endocarditis**, to which the right heart is the more predisposed during uterine life, because it receives the blood arterialized in the placenta, and to a certain degree assumes the functions later taken up by the left ventricle.

Symptoms and Diagnosis.—Congenital lesions of the heart may be unattended with symptoms—*latent congenital heart-disease.* Among the appreciable manifestations are **congenital cyanosis** and **local cardiac alterations.**

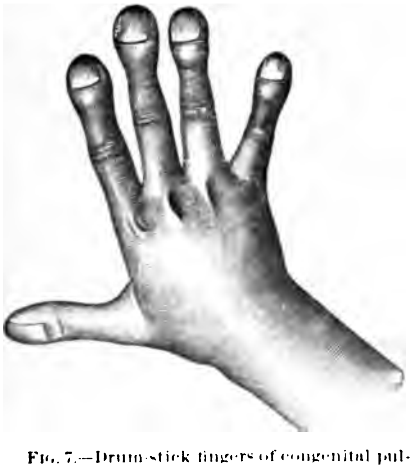


FIG. 7.—Drum-stick fingers of congenital pulmonary stenosis (from a photograph (personal observation, Zurich clinic)).

Congenital cyanosis is characterized by a bluish-red, at times almost blackish-blue, discoloration of the skin and mucous membranes, so that the condition has been named *morbus cæruleus.* Obviously the appearance is the result of deficient oxidation of the blood; possibly, however, the unusually large number of red blood-corpuscles and the large amount of hemoglobin also may contribute to the result. Not rarely children with congenital lesions of the heart are *apparently dead* at birth. If resuscitation take place, the peculiar color of the skin is often conspicuous from the beginning. In other instances the cyanosis appears only on crying, on exposure to cold, and after physical exertion. The lips and the nose appear swollen and the eyeballs frequently project markedly forward. The terminal phalanges of the fingers and toes exhibit peculiar alterations, being expanded like drum-sticks—*drum-stick fingers* (Fig. 7). At the same time there is a tendency to frost-bite and inflammation of the terminal phalanges. The skin feels cool, and the patients suffer readily from cold and prefer the neighborhood of the stove. They avoid play with those of their own age, as they suffer from palpitation of the heart and shortness of breath, and they appear quiet, retiring, introspective, and mentally unresponsive.

The *local cardiac alterations* depend upon the nature of the congenital heart-lesion. In the presence of congenital pulmonary stenosis a systolic murmur is audible over the pulmonary orifice, and dilatation and hypertrophy of the right ventricle take place. Patulousness of the foramen ovale and deficiency of the inter-

r the interventricular septum can never be recognized
nty. At times extended presystolic or systolic mur-
udible over the heart. Changes in the duct of Botal
to been insusceptible of diagnosis.

icipal dangers from congenital lesions of the heart con-
ness of the myocardium and stasis, and in the case of
pulmonary stenosis in *pulmonary tuberculosis*. Most
congenital heart-disease do not survive the fourteenth

sis.—The prognosis is **unfavorable** in accordance with
en said. Recovery is out of the question.

ent.—The treatment is the same as that of acquired
ie heart.

AMMATION OF THE ENDOCARDIUM (ENDOCARDITIS).

orms of endocarditis have been distinguished, and
een designated *ulcerative*, *verrucose*, and *contracting*.
nd verrucose endocarditis are due to the activity of
the same microörganisms may give rise to the one or
iety. Transitional and mixed forms are also observed
ically and clinically. Contracting endocarditis leads
, contraction, and frequently also to calcification of
ium, and is often the sequel of a preëxisting verru-
litis. In other instances it develops independently
ition of senility, and often in association with arterio-
cessive physical activity, excessive use of alcohol,
e of the kidneys, gout, and diabetes also are causes.
inction from ulcerative and verrucose endocarditis,
g variety pursues a *chronic course*, so that it has been
o *chronic endocarditis*. It constitutes the most com-
cause for *valvular lesions of the heart*, and it exhibits
ncy to become again acute, when it is designated
racting endocarditis.

ULCERATIVE ENDOCARDITIS.

—Ulcerative endocarditis is most commonly the
reëxisting **wound-infection**. Then, it is not rare in
f **infectious diseases**. At times a causative factor is
and under such conditions the designation *crypto-*
rditis has been employed. In this category prob-
so the instances in which **exposure to cold, excessive**
ty, and **psychic disturbances** have been assigned as
s.

d-infections it should especially be mentioned that ulcer-
is not rarely develops in association with *puerperal fever*.

Further, the smallest wound is sufficient to afford entrance for bacteria into the blood and to the endocardium. In this connection, not only external, but also internal, wounds must be taken into consideration; for instance, those of the mucous membrane of the digestive apparatus. Among *infectious diseases acute articular rheumatism* especially is a frequent cause of ulcerative endocarditis. Among the acute exanthemata, *scarlet fever* is of especial importance. Of late ulcerative endocarditis has been observed a number of times in the sequence of *gonorrhea*. In cases of *cryptogenetic ulcerative endocarditis* the tonsils may often be the portal of entry for the bacteria.

Experience has shown that *women are more commonly attacked* by ulcerative endocarditis than men. The disease is rare in childhood. It occurs most commonly between the twentieth and the fortieth year of life.

Anatomic Alterations.—The first changes in ulcerative endocarditis consist in a *deposit* upon the endocardium that is composed principally of masses of bacteria. If this be detached, **a loss of tissue**, usually sharply circumscribed, becomes apparent. Frequently fibrinous masses are deposited at these points (**endocarditic vegetations**), which, as a rule, are less abundant than in cases of verrucose endocarditis. The lesion of the endocardium may lead to perforation and destruction of the valve-structure; or it may give rise to an **acute aneurysm of the valve** if only one layer of the valve is destroyed. The ulcerative process may also extend to the myocardium and give rise to an **acute myocardial ulcer.** Should saccular dilatation of the myocardium take place at such a point, an **acute aneurysm of the heart** will result.

Ulcerative endocarditis most frequently attacks the valves of the heart, especially those of the left side, and the alterations involve the lines of closure of the valves. It attacks the tendinous cords, the papillary muscles, and the mural endocardium much less commonly. At times several valves are involved simultaneously, both upon the right and upon the left side.

The most frequent exciting agent of ulcerative endocarditis is the staphylococcus pyogenes aureus and the streptococcus pyogenes, although a number of other bacteria have been isolated from the endocarditic inflammatory areas, at times specific bacteria, such as gonococci and typhoid-bacilli.

Ulcerative endocarditis is characterized by a great tendency to the development of *bacterial embolism*. Small masses of bacteria become detached from the inflamed endocardium, and are carried through the arterial blood-current into the most diverse organs, where they become lodged in the smaller blood-vessels, forming at first a grayish or grayish-white center, surrounded by a zone of hyperemia. Soon, however, inflammation and suppuration are superadded. Frequently the individual organs contain numerous emboli and foci of suppuration, and only few viscera escape this complication.

toms and Diagnosis.—Ulcerative endocarditis is only
of diagnosis when in addition to a **septic general condi-**
f a valvular lesion of the heart are also present. As long
iac valve remains functionally competent only the general
can be recognized. This at times pursues a course sug-
typhoid fever (continued fever, dry, coated tongue, dis-
lomen, roseola, splenic enlargement, diarrhea); at other
ls followed by elevation of temperature, lasting for
rs, and recurring with a certain degree of regularity,
enlargement, raise a suspicion of *intermittent fever;* or
purative inflammation occurs in one or more organs,
ther with possible chills, are due to bacterial emboli.
tful cases it is especially important to look for *embolic*
in the skin, the mucous membranes (of the mouth, the
), and the retina. In these structures hemorrhages may
ly observed, in which the bacterial plug may be recog-
white center. Further, roseolous, scarlatiniform, and
ptions also occur upon the skin.

e of doubtful diagnosis a physician trained in bacteriologic
ld examine the stools, the urine, and the blood of the rose-spots
ce of typhoid-bacilli, or the blood for malarial plasmodia. In
ative endocarditis, on the other hand, it is not uncommonly
tain pyogenic cocci from the blood.

vular alterations as may be present will manifest them-
docardial murmurs, which are mostly systolic and less
liastolic in occurrence. To these dilatation of the
e superadded. The disease usually pursues too rapid
hypertrophy to develop.
y take place within three days. At times, however,
s protracted for months. Recovery is scarcely to be
Life is terminated either with progressive failure of
in consequence of secondary suppuration, or of embo-
nportant cerebral vessel.
is.—The prognosis is almost unexceptionably un-

nt.—A **nutritious liquid diet** should be prescribed
n with egg, coffee, tea, wine), an **ice-bag** is kept ap-
ously to the precordium, and **digitalis** is prescribed to
d slow the action of the heart; the last may be com-
enzoic acid and camphor when asthma is present.
rid and **quinin** have also been employed for the pur-
oying the bacteria upon the endocardium.

VERRUCOSE ENDOCARDITIS.

.—Verrucose endocarditis is caused by the same bac-
cerative variety. Why in the one instance the disease
errucose type and in the other the ulcerative type is

not definitely known. **Infectious diseases** of all kinds are the most common cause of the disorder. Among the most conspicuous of these are *acute articular rheumatism* and in childhood *scarlet fever*. **Debilitating diseases** (carcinoma, pulmonary tuberculosis, chronic nephritis) also favor infection and inflammation of the endocardium. **Exposure to cold** and **injury** further act as etiologic factors, although these must be considered only as *contributory causes* favoring infection of the endocardium. Experience has shown that verrucose endocarditis occurs *more commonly in men* than in women, and especially *between the twentieth and the fortieth year of life.*

Anatomic Alterations.—The characteristic alterations of verrucose endocarditis consist in thrombotic deposits, **endocarditic vegetations,** upon the inflamed areas of the endocardium, which not rarely in appearance resemble an extensive cock's comb, cauliflower, or condyloma. As a rule, the endocarditis involves the valves, the vegetations being situated at the lines of closure. The mitral valve is attacked with especial frequency. Inflammation of the pulmonary and tricuspid valves is uncommon. As may be understood, the vegetations are capable of interfering with the function of the affected valves and inducing the manifestations of a valvular lesion of the heart. In the further course of the morbid process organization of the vegetations takes place. Thickenings occur, even calcification of the valve, with contraction and adhesions, and the valvular lesion becomes a permanent one.

Bacteria can frequently be demonstrated in the vegetations only by culture-methods. When the vegetations have existed for some time bacteria are no longer present.

A frequent complication of verrucose endocarditis is constituted by **emboli,** which result from the disintegration of endocarditic vegetations. These do not, as a rule, excite inflammation in the affected organ, because they do not contain bacteria or contain them in too small number, but they give rise to the manifestations of an embolic infarct.

Symptoms and Diagnosis.—Verrucose endocarditis can be recognized only from the development, in the course of days or weeks, of the **symptoms of a valvular lesion of the heart,** most frequently those of mitral insufficiency. In the condition last named a systolic murmur first appears. Gradually dilatation of the right side of the heart is superadded, and finally the pulmonary second sound becomes accentuated and indicates hypertrophy of the right heart. At times **elevation of temperature, palpitation of the heart, a sense of constriction,** and **irregularity of the pulse** appear; but frequently the disease develops quite insidiously, and is recognized only by thorough and repeated examination of the heart. If *embolism* develops, the symptoms described on pages 42 and 43 make their appearance.

osis.—If verrucose endocarditis has given rise to a
sion of the heart, this usually persists for the remainder
l is attended with all of the dangers that have already
:ioned, especially derangements of compensation and

nent.—The treatment is the same as that for ulcerative
is (page 49).

THROMBOSIS OF THE HEART.

r.—Fibrinous deposits upon the endocardium take place when
ium is diseased, and in consequence has lost its capability of
the fluidity of the blood in the cavities of the heart. This
njunction with inflammations of the endocardium and with
:es, so that a distinction can be made between *inflammatory* and
mbosis of the heart. Slowing of the blood-current favors the
thrombi. Whether a morbid tendency of the blood to the
fibrin occurs or not is doubtful.

e Alterations.—Cardiac thrombi form, besides, upon inflamed
especial frequency near the apex of the heart, in the ventricles,
ricular appendages. They vary in size and form. At times
masses are lodged between the fleshy columns, occupying their
ore or less completely; at other times the thrombi are sus-
lender pedicles in the cavity of the heart—so-called *true cardiac*
ncommon variety of cardiac thrombus is the *globular thrombus*,
able thrombus of roundish shape in the auricle, which may
iate death by sudden closure of a venous orifice. At times
rgo softening in their interior, and then they may contain a
wn or pus-like fluid. Cardiac thrombi are differentiated from
fibrinous deposits by the fact that they are tougher, rather
or grayish-red, and adherent to the endocardium.

s and Diagnosis.—The symptoms of cardiac thrombosis are
e nature that even under the most favorable conditions the
only be suspected with a certain degree of probability. At
manifestations are entirely wanting—*latent cardiac thrombosis.*
nces signs of *weakness of the heart* become more and more
n still other instances fragments of thrombus become broken
heir way as *emboli* into the distribution of the pulmonary
e aorta, respectively. *Sudden death* is possible as a result of
ardiac orifices. At times signs of *constriction of a cardiac*
when a thrombus merely obstructs but does not entirely
ifice.

—The prognosis is more favorable in cases of inflammatory
f marantic cardiac thrombi, for in the former the condition
nvolution to a certain degree, whereas in the latter there is
eased growth.

.—The treatment is purely symptomatic, and should be
t individual prominent symptoms.

III. DISEASES OF THE PERICARDIUM.

INFLAMMATION OF THE PERICARDIUM (PERICARDITIS).

Etiology.—Like most inflammations in human beings, inflammation of the pericardium also probably results only when *bacteria* capable of exciting inflammation have gained access to the pericardium. In addition to the usual exciting agents of suppuration (Streptococcus pyogenes, Staphylococcus pyogenes aureus and albus) specific bacteria, especially tubercle-bacilli, also act as etiologic factors. The agencies otherwise mentioned as causes of the disorder have only the significance of *contributory and accessory influences for the infection.*

Inflammation of the pericardium occurs most frequently in the sequence of **infectious diseases.** The most prominent position in this connection is occupied by *acute articular rheumatism,* which not rarely gives rise at the same time to endocarditis and pericarditis, and even also to myocarditis. **Exposure to cold, injuries, and debilitating diseases** (chronic nephritis, carcinoma, long-continued suppuration, scorbutus, purpura, leukemia) likewise favor infection of the pericardium. At times the condition arises by extension from **contiguous inflammation.** Pericarditis is associated with especial frequency with left-sided pleurisy. In some instances pericarditis occurs after **preexisting disease of the pericardium** of other nature; thus after carcinoma, sarcoma, tuberculosis, and gumma. At times no cause whatever is elicitable—*spontaneous pericarditis.* Under such conditions the disease is often tuberculous. The infection is engendered with especial frequency by tuberculosis of the tracheobronchial lymphatic glands. Experience has shown that inflammation of the pericardium occurs most frequently in *men,* and at the *best period of life.*

Anatomic Alterations.—Accordingly as pericarditis is attended with the development of a fibrinous or a fluid exudate, a distinction is made between *pericarditis sicca* (*dry pericarditis*) and *pericarditis humida* (*pericarditis with effusion*).

In *pericarditis sicca s. fibrinosa* (*dry* or *fibrinous pericarditis*) the surface of the pericardium is at first found to be dull, as if moistened with vapor and covered with a thin, veil-like fibrinous membrane, which can be detached with a knife. Beneath this the pericardial tissue appears reddened. The fibrinous membrane gradually becomes thicker, more opaque, more deeply yellow, and in consequence of the movements of the layers of the pericardium upon one another the surface acquires an irregular, rough, and papillary appearance, by reason of which the terms *villous heart,*

m (*cor villosum s. tomentosum*) have been employed. The
of such a heart has also been compared with that of a
a honeycomb, of the fourth stomach of ruminants, or of
es of buttered bread that have been separated. As
rinous pericarditis involves simultaneously both peri-
d epicardium. Only rarely is one of the layers of the
n alone inflamed. In accordance with the extent of
mation a *circumscribed* (*local*) and a *diffuse* (*total*) *peri-*
distinguished.

ditis humida (*pericarditis with effusion*) usually develops
inous pericarditis, the formation of a fluid exudate being
to the deposition of fibrinous coagula. If the inflam-
d is deficient in cells and still to a certain degree trans-
condition is described as *serous pericarditis* or, more
serofibrinous pericarditis. Under other conditions the
embles the pus of an ordinary abscess—*purulent peri-*
yopericardium. Finally, the effusion may be bloody—
pericarditis. At times, in consequence of the presence
tive bacteria, putrid decomposition takes place in a
usion—*putrid pericarditis.* This occurs especially as
septic influences; for instance, puerperal fever. The
luid exudate may exceed three liters, so that the peri-
pears tensely distended, and when punctured the fluid
a stream. It is worthy of note that the heart-muscle,
its weight, sinks in the exudate, and that especially
contiguous to the pericardium exhibit inflammatory
Microscopically such portions are found to participate
nmatory process.

tis but rarely undergoes recovery without leaving
n the pericardium. Of subordinate importance are
ickenings of the epicardium—*tendinous spots, maculæ*
bidæ, also designated insulæ. Frequently connective-
ons take place between the two layers of pericardium
synechiæ or *adhesions*). These sometimes cause com-
tion of the pericardial cavity—*obliteratio s. concretio*
At times the synechiæ contain inspissated, cheesy,
remnants of the fluid exudate. In rare cases almost
art appears to be enclosed in a calcareous capsule.
inflammation extends from the pericardial cavity to
ace of the pericardium—*external pericarditis.* Under
ns also connective-tissue adhesions may form in the
me, and these may surround the large arteries and
runks of the heart, and possibly bind the pericar-
sternum and to the vertebral column. Such altera-
een designated *indurative* or *fibrous mediastinoperi-*

s.—Pericarditis can be recognized only from the *physical alterations in the heart.* Constitutional symptoms are wanting or are only of indefinite nature.

Fibrinous pericarditis is disclosed by the occurrence of a **pericarditic friction-murmur.** This is characterized by its superficial, rasping character, by not being confined strictly to the cardiac phases, and by usually exhibiting numerous interruptions which are dependent upon the movements of the auricles and the ventricles. It is at times so loud that it can be felt with the hand as *pericardial friction* or *affrictus pericardiacus,* and that the patient may himself be conscious of its presence in the thorax as a rubbing. Pericardial friction-murmurs are usually confined to the area of cardiac dulness, or they are diffused in any event for but a short distance beyond. They are intensified by the pressure of the stethoscope. Their intensity varies also with the position of the body, and is increased as a rule by over-inclination forward. Frequently pericardial friction-murmurs are audible for days and weeks; at times, however, for only a few hours, because the inflammation is sometimes of but short duration.

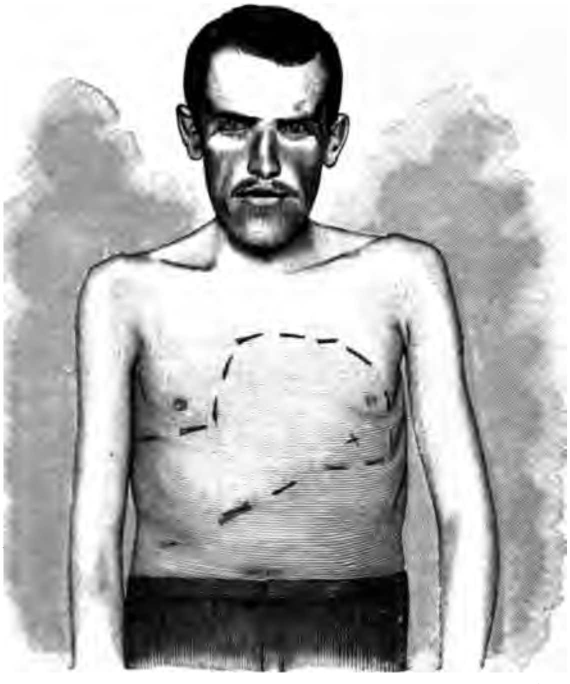


FIG. 8.—Area of cardiac percussion-dulness in a case of pericarditis with effusion: X, situation of the apex-beat. From a photograph (personal observation, Zurich clinic).

Pericarditis with fluid effusion first leads to increase in size and

shape of the area of cardiac dulness. This is induced
on of the pericardium by the fluid. The area of car-
s is increased both to the right and the left, and from
nward, and at the same time it assumes the shape of a
Fig. 8).

diagnosis it is important that frequently the area of
ness extends to the left beyond the apex-beat, because
n the pericardial cavity is distributed to the right and
yond the borders of the heart. **Gradual disappearance**
-beat in the course of the disease is likewise a valuable
e layers of exudate gradually increasing in thickness
d upon the anterior surface of the heart, and the heart-
omes the more deeply submerged. In consequence
n the intensity of the heart-sounds also takes place.

mptoms of more subordinate significance may be mentioned
ng of the precordium in consequence of pressure exerted by
al exudate and inflammatory paresis of the intercostal mus-
neous edema in the precordium, the latter resulting from the
ma.

najority of cases pericardial friction-murmurs are en-
n addition to signs of accumulation of fluid in the
, as usually besides fluid effusion fibrinous inflammatory
e present. Friction-murmurs are absent only when
d effusions have been poured out, because then a
on each other of the layers of the pericardium is
Such murmurs appear only when a portion of the
e has undergone absorption.

onal symptoms may be entirely wanting in cases of
of the pericardium. Frequently irregular febrile
present. Not rarely the patients complain of a sense
on, of pressure, and even of pain in the precordium.
f the heart appears accelerated and irregular, and the
is conspicuous for its slight tension.

tion of inflammation of the pericardium may extend
months.

he most frequent and most serious *complications* is
eakness, with symptoms of *stasis,* and a considerable
patients die in consequence of increasing *edema.*
lysis of the heart, for instance from carelessness in
sitting or the erect posture, is less common. At
stations due to pressure of the pericardial exudate
ring organs occur. Thus, there may be unilateral
eral paralysis of the recurrent laryngeal nerve, con-
the esophagus, obstinate singultus, and vomiting.
e lower lobe of the left lung is compressed until
is expelled, with the development of dulness on
creased vocal fremitus, and bronchial breathing. At

times the inflammation of the pericardium extends to the pleura, and then *pleurisy* is superadded, usually left-sided. Not rarely attacks of *accelerated heart-action* and *dyspnea* occur which may be excited by physical or mental exertion, but which appear also during states of profound rest.

Rupture of a pericardial effusion, with the formation of an external or an internal *pericardial fistula*, may be mentioned as an unusually rare complication.

When pericarditis terminates in recovery a morbid *hyperirritability of the myocardium* often persists for a considerable time, and which is manifested in a marked tendency to accelerated and irregular cardiac action. Among the residua of pericarditis *tendinous spots* are unattended with symptoms; in only one instance have I been able to detect friction-murmurs over an especially dense and thick tendinous spot. *Pericardial adhesions* also are unattended with symptoms so long as they do not affect the circulation in the coronary arteries of the heart and the movements of the heart-muscle. If the coronary arteries are constricted by adhesions, the nutrition of the myocardium must suffer, and signs of *cardiac weakness* and *stasis* appear, to which the patient finally succumbs. Adhesions that interfere with the systolic movement of the heart downward give rise to *systolic retraction in the region of the apex-beat* with the systolic shortening of the heart. This becomes especially marked, and involves also the contiguous thoracic wall, when the apex of the heart is adherent to the inner aspect of the chest, and is capable of exerting direct traction on the chest-wall. *Indurative mediastinopericarditis* is indicated by an *intermittent inspiratory* or *paradoxic pulse* and *inspiratory distention of the cervical veins*. Naturally, these are not unequivocal symptoms, for similar manifestations are sometimes observed also in association with pericarditis, pleurisy, and aneurysm of the aorta. Both the diminution in size and even the disappearance of the pulse during inspiration and the synchronous increased distention of the cervical veins may be explained by the fact that the aorta and the vena cava are surrounded by adhesions, and their lumen becomes constricted by traction during inspiration.

Diagnosis. Whenever pericardial *friction-murmurs* are heard fibrinous pericarditis may be diagnosed, for it is extremely rare for hemorrhages, excessive dryness (in cases of Asiatic cholera) or tendinous patches of the pericardium, to give rise to such murmurs. Pericardial murmurs may be confounded with endocardial and pleuropericardial external pericardial murmurs.

[illegible] pericardial murmurs [illegible] audible beyond [illegible] confined to [illegible] of inter- [illegible] endocardial murmurs,

ıter, but louder, because the dilating lung exerts pressure upon
ɔf the pericardium and favors their rubbing upon each other.
ılso be increased by pressure with the stethoscope, and they vary
ntensity with the posture of the body.
ricardial friction-murmurs occur with an intact pericardium
ous pleurisy has developed in close proximity to the pericar-
ıat the movements of the heart give rise to pleural friction-
The dependence of doubtful murmurs upon respiration may,
ually be demonstrated by deep inspirations. If the breath be
ep inspiration, the pericardial influences are suspended, and in
do not appear when the breath is held.

presence of pericarditis with effusion there is a possi-
ɔnfounding the enlarged area of cardiac percussion-dul-
ilatation of the heart. It is then important to determine
e area of cardiac dulness extends to the left beyond the
which is indicative of the presence of fluid in the peri-
ıd whether the apex-beat gradually disappears in the
he disease. When pericarditis with effusion is present
ounds become faint and the action of the heart is
at all, visible. Gerhardt has further called attention
lacement upward of the upper limit of cardiac dulness
ıg posture in cases of pericarditis.

pleuritic effusions and *infiltration of the median borders of the*
re rise to increase in the area of cardiac dulness and to errors
but the dulness then usually assumes an irregular shape, and
ion of the lung is present bronchial breathing is audible.

racter of the effusion can be determined with certainty
ɔloratory puncture of the pericardium; otherwise de-
ust be placed upon the results of clinical experience,
that acute articular rheumatism is usually attended
rinous, septic processes with purulent, and scorbutus,
, sarcoma, and often also nephritis, with hemorrhagic

sis.—Pericarditis is under all circumstances a serious
en the cases that at first appear to be mild may with
are acquire a grave character. Of great importance
n to the prognosis is the *etiology.* While pericarditis
nce of acute articular rheumatism is usually followed
the prognosis when septic factors are operative is
ıore serious, and in the presence of new-formations it
unfavorable.

ent.—All varieties of pericarditis require the same
ment (rest in bed, easily digestible diet, especially milk
Further than this the treatment will vary with the
the exudate. In cases of *fibrinous pericarditis* an
d be applied continuously to the precordium, and if
r marked dyspnea be present an injection of morphin

℞	Morphin hydrochlorate	0.3 (4½ grains);
	Glycerin,	
	Distilled water,	each 5.0 (75 minims).—M.

Dose: 0.5 c.c. (8 minims) subcutaneously.

If the action of the heart be accelerated, enfeebled, and irregular, digitalis should be prescribed:

℞	Powdered digitalis-leaves,	0.1 (1½ grains);
	Diuretin,	1.0 (15 grains);
	Sugar,	0.5 (7½ grains).—M.

Make ten such powders.
Dose: 1 powder every three hours.

In cases of *serous pericarditis* also a similar course of treatment may be recommended. From sorbefacients (potassium iodid internally, potassium-iodid ointment or iodoform-ointment by inunction, and tincture of iodin applied locally) no great measure of success is to be expected. At times repeated application of an ordinary **cantharidal plaster** will hasten the absorption of the effusion. If absorption fails to take place after the lapse of weeks, or if perhaps after the fourth week the amount of effusion increases, or if it is from the outset so extensive that death is threatened through interference with the action of the heart, artificial evacuation of the effusion by **puncture of the pericardium** may be recommended.

Purulent pericardial effusions should be evacuated immediately by **opening the pericardium** with a knife, because experience has taught that absorption cannot be effected by any means, and if delay be too much prolonged irremediable cardiac weakness will develop. The same statement applies to *putrid pericarditis*. *Hemorrhagic pericarditis* has been observed to assume such enormous proportions in cases of scorbutus that puncture of the pericardium was necessitated.

PNEUMOPERICARDIUM.

Anatomic Alterations.—Pneumopericardium signifies an *accumulation of gas in the pericardial cavity*. If the amount of gas be considerable, the pericardium is tensely distended, and when punctured the gas escapes with a hissing sound. As a rule, pure pneumopericardium is not encountered, but a *hydropneumopericardium*; that is, in addition to gas fluid also is present in the pericardial cavity. Accordingly as this fluid is serum, pus, or blood, a distinction is made between *seropneumopericardium*, *pyopneumopericardium*, and *hemopneumopericardium*. The pericardium at the same time presents evidences of inflammation.

Etiology.—Pneumopericardium is a rare disease. It develops when the *pericardial cavity communicates with air-containing viscera* or through the thoracic wall directly with the *external air*. Such occurrences have been observed in cases of ruptured pulmonary cavities, carcinoma of the esophagus, gastric ulceration, carcinoma of the stomach, rupture of purulent pericardial effusions into air-containing viscera, or externally, injuries of the chest-wall, and the like.

It has been maintained that a pyopneumopericardium may be developed

pericardium *by decomposition of the pus.* This possibility must be
s *gas-generating bacteria* are known to exist.

ms and Diagnosis.—The *symptoms of pneumopericardium* consist
rance of the area of cardiac dulness in the dorsal decubitus, and
e instead of *signs of a smooth-walled cavity: a metallic percussion-*
is brought out with especial distinctness when auscultatory per-
practised with the aid of a plexor and pleximeter. The *heart-*
nsequence of the resonance also seem to possess a metallic char-
a bell. If the air-fistula be patulous, percussion further will
ked-pot sound. In the sitting posture, and on bending forward,
ill become again applied to the chest-wall, and can then be
d by the dulness on percussion.

rdial splashing-sound is distinctive of *hydropneumopericardium*
murmur). This is developed by the movement to and fro of the
s of the pericardium through the action of the heart. At times it
d at some distance from the patient. In addition *changes in the*
enomena take place in accordance with the position of the body.
ependent upon the fact that the fluid always seeks the lowest
ver it dulness exists, while the gas-generating cavity yields a
on percussion.

with pneumopericardium and hydropneumopericardium com-
of a sense of constriction and even of pain in the precordium,
ion of the heart is accelerated and often irregular; they suffer
and threatening weakness of the heart, because the free move-
organ is prevented. *Weakness of the heart-muscle* and *paralysis*
re the principal dangers of the disease.

s.—The prognosis is unfavorable because of the serious nature
y disorder, independently of the dangers that attend pneumo-
and hydropneumopericardium themselves.

nt.—In cases of *pneumopericardium* and *hydropneumopericar-*
should first be made to allay fear and dyspnea by means of a
njection of morphin. Should the collection of gas be consider-
cardium may be punctured by means of a hollow needle, whose
y is covered with a rubber tube, the other end of which is
h the surface of a solution of carbolic acid (5 : 100). In cases
pericardium the pericardium should be opened by incision,
es of seropneumopericardium and hemopneumopericardium
e pericardium, with subsequent treatment as in cases of serous
gic pericarditis will suffice.

DROPSY OF THE PERICARDIUM (HYDROPERICARDIUM).

Alterations.—The accumulation of a *serous transudate* in the
vity is known as hydropericardium. The pericardium always
tain amount of serous fluid, so that hydropericardium is only
n the pericardial cavity contains more than 100 c.c. of clear,
fluid. The amount, however, may reach several liters. Not
cardial tissue appears edematous, and the myocardium pale
with fluid. Hydropericardium is distinguished from a serous
absence of flocculi and the clear appearance of the fluid.

—**Transudation** into the pericardial cavity results from causes
se that bring about transudation into other serous cavities
ubcutaneous connective tissue, with which it is thus usually
iation. The condition present, therefore, is usually one of
increase of pressure in the venous system. It is observed quite
ssociation with diseases of long standing, such as pulmonary

tuberculosis, carcinoma, suppuration, nephritis, respiratory and cardiac disease. Rarely, local causes of stasis are present in the coronary veins, in consequence of adhesions or tumors of the pericardium.

Symptoms and Diagnosis.—Dropsy of the pericardium gives rise to the same *local cardiac alterations* as a collection of pericardial fluid exudate, except that a pericardial friction-murmur is wanting. The area of *cardiac dulness* is increased in extent, and projects beyond the apex-beat outward toward the left, the apex-beat becomes feebler and finally disappears, and the heart-sounds are faint. In addition there are signs of *weakness of the heart,* which is induced in part by the primary disorder, and in part by interference with the action of the heart. Usually there is complaint of marked dyspnea and a sense of constriction, because the movement of the lungs and of the diaphragm is embarrassed by coexisting hydrothorax and ascites. Death in consequence of *paralysis of the heart* is the most frequent termination.

Prognosis.—Dropsy of the pericardium is not unjustly in evil repute among the laity, for frequently it is associated with incurable conditions, and, besides, the disorder itself is attended with the danger of cardiac weakness and paralysis of the heart. Recovery is, however, possible if the cause can be removed.

Treatment.—Treatment should in the first place be *causal.* Symptomatically resort will often be had especially to *heart-tonics,* and particularly to digitalis and diuretin. Extensive transudates require *puncture of the pericardium.*

HEMOPERICARDIUM.

Accumulations of blood in the pericardial cavity occur especially in association with *injuries and rupture of the heart and of aneurysms of the aorta, pulmonary artery, or coronary arteries.* Their recognition depends upon the fact that the area of *cardiac percussion-dulness* increases rapidly in consequence of the increasing accumulation of blood, while the apex-beat disappears and the sounds of the heart become fainter, and at the same time *manifestations of internal hemorrhage* appear. Death may result from *hemorrhage* or in consequence of *paralysis of the heart.* The prognosis is, therefore, always serious. Therapeutically *stimulants* may be recommended; for instance, camphorated oil (a syringeful subcutaneously every two to four hours). Not much will be accomplished by the use of *hemostatics* (ice-bag, subcutaneous injection of extract of ergot, fluid extract of hydrastis, twenty-five drops thrice daily, etc.). I have averted death in one case by *puncture of the pericardial cavity* and aspiration of the blood.

CHYLOPERICARDIUM.

In rare instances lacteal vessels rupture and an accumulation of chyle takes place in the pericardial cavity, which, if in sufficient amount, may give rise to an increase in the area of cardiac percussion-dulness.

TUMORS OF THE PERICARDIUM (NEOPLASMATA PERICARDII).

Tumors of the pericardium (carcinoma, sarcoma, fibroma, enchondroma, lymphoma) are rare. They are often unrecognized during life. Carcinomata and sarcomata, which are almost always secondary, are likely to be associated with pericarditis with effusion, and this is frequently hemorrhagic. This latter condition may be recognized; but, as a rule, it is only learned at autopsy that the new-formations in the pericardium were the actual cause.

IV. CARDIAC NEUROSES.

PAROXYSMAL TACHYCARDIA.

oms and Diagnosis.—Paroxysmal tachycardia is
ed by **attacks of accelerated cardiac activity.** These
occur without demonstrable cause, while at other times
v upon physical or mental exertion, gastric derange-
use of coffee, tea, alcohol, and the like. The number
ats may exceed two hundred in the minute, and they
w one another so precipitately that each contraction
rt is not sufficient to induce a palpable pulse in the
ry. Not rarely there is also irregularity in the action
rt.

graphic observations usually disclose *alterations in the pulse-*
nsequence of reduction in tension the pulse is frequently mono-
crotic.

le, **local cardiac alterations** are demonstrable. The
is shaken and raised with unusual vigor, and through-
eased extent, and the systolic sound of the heart at
res a clattering character, which has been designated
étallique, and has been attributed to vibrations of the
At times dilatation of the right ventricle takes place.
ctive pulsation of the carotids in the neck is observed.
subjective disturbances the distressing sense of ham-
beating in the chest predominates. Besides, a sense
nstriction, shortness of breath, and accelerated breath-
ticeable. Not rarely pallor, cyanosis, and marked
ntion are observed.
k may *terminate* quite suddenly. At times its termi-
receded by eructation, vomiting, discharge of flatus,
ntents, or pale, watery urine. The *duration* of an attack
tween a few minutes and several days. While some
subjected to only a single attack or but a few attacks,
have several attacks daily for years, and at times
he whole of life, so that the disorder may become a
affliction. A fatal termination is rare, but may occur
nce of paralysis of the heart, enfeeblement of the
, and stasis or cerebral hemorrhage.
nition of the disease is easy from its paroxysmal occur-
ich it is distinguished from the accelerated activity
that develops when unusual resistance is interposed
l blood-current, as, for instance, with uncompensated
ns of the heart.

It is noteworthy that some persons complain of all of the symptoms of paroxysmal tachycardia without the action of the heart being at all accelerated. For this reason a distinction has been made between *subjective* and *objective palpitation of the heart.*

It is important in every case to determine the *cause of the disorder*, because the prognosis and treatment are dependent upon it.

Acceleration of the action of the heart may take place in consequence of *paralysis of the vagus* or of *irritation of the sympathetic*. The former appears to occur the more frequently, and may be assumed to exist when pressure upon the vagus in the neck speedily brings the attack to an end. On the other hand, in cases of tachycardia due to irritation of the sympathetic morphin will often cause speedy subsidence of the attack. A positive differentiation, however, can frequently not be made.

Etiology.—Persons with a *readily irritable nervous system* are especially predisposed to paroxysmal tachycardia. Among the actual causes are at times **diseases of the nerves**, and at other times reflex irritation. Not rarely the disorder develops in the course of certain *central neuroses*, particularly hysteria, neurasthenia, and hypochondriasis. **Psychic disturbances** also play an important etiologic rôle; for instance, joy, fright, grief, fear, embarrassment, nostalgia, and disappointment in love. At times **toxic influences** may be operative, such as excessive use of alcohol, tobacco, coffee, and tea. The palpitation of gouty patients possibly also belongs in this category. At times paroxysmal tachycardia develops in connection with states of **exhaustion of the nervous system**; for example, after severe typhoid fever, loss of sleep, and sexual excesses. **Anatomic alterations in the central nervous system** (hemorrhage, abscess, tumor of the brain, tabes dorsalis, multiple sclerosis, compression of the vagus by tumors) may also be considered as causes of paroxysmal tachycardia. The disorder occurs with especial frequency in consequence of **reflex irritation**. Thus gastric, intestinal, hepatic, renal, uterine, and ovarian disease may be followed by paroxysmal tachycardia, as may also valvular disease of the heart, disease of the myocardium, of the pericardium, of the nasal turbinate bones, and even of the teeth. Nevertheless, there are still cases in which no cause can be demonstrated. The disorder occurs more frequently in *women* than in men, because in the former the nervous system is more irritable, and diseases of the female generative organs constitute a not uncommon point of origin. Although the affection is not unknown among children also, it occurs much more frequently at about the period of puberty.

Anatomic Alterations.—Distinctive anatomic alterations are not known. Dilatation, fatty degeneration, and relaxation of the heart are secondary manifestations. Stix reported the occurrence in one case of degeneration of the fibers of the vagus from compression by bronchial glands.

Prognosis.—The prognosis is favorable only in such cases in which the causative condition is curable. Life is, naturally, not endangered as a rule.

Treatment.—During the *paroxysm of palpitation of the heart* it is advisable for the patient to seek a quiet, slightly darkened room, to occupy the horizontal posture, to apply an ice-bag to the precordium, and to swallow small bits of ice, or water-ice. Some patients have learned how to obtain relief with the aid of domestic remedies, for instance by means of a cup of black coffee; while others obtain speedy relief from pressure on and irritation of the vagus in the neck, or from pressure upon certain points on the abdomen. Usually an attack can be speedily terminated by means of a subcutaneous injection of morphin:

℞ Morphin hydrochlorate, 0.3 (4½ grains);
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: From 0.25 to 0.5 c.c. (4 to 8 minims) subcutaneously.

To *prevent subsequent attacks,* the causes of the disorder must be kept in view, and naturally the most varied measures and modes of treatment will be taken into consideration.

PAROXYSMAL BRADYCARDIA.

Paroxysmal bradycardia is characterized by *attacks of unusual infrequency of cardiac action,* so that the pulse-beat may be less than thirty in the minute. In addition there may be a sense of oppression in the precordium, of fear, vertigo, and profound syncope. I have observed such conditions most frequently in the sequence of *infectious diseases,* especially after acute articular rheumatism and typhoid fever; but they may be due also to *toxic influences* (coffee, tea, tobacco), and to *fat heart, chronic myocarditis,* and *sclerosis of the coronary arteries.* The condition should not be confounded with the persistent slowing of the action of the heart observed in connection with icterus, intestinal diseases, and the puerperium. The *horizontal posture* and *stimulants* (ethereal tincture of valerian, 30 drops; camphorated oil subcutaneously) are best adapted to the relief of such symptoms as may be present.

CARDIAC INTERMITTENCY.

Cardiac intermittency is characterized by attacks in which the action of the heart is suspended for a number of seconds at a time, to be resumed again in a regular manner. During the attack the patients complain of a sense of oppression, of fear, and of a premonition of death. The affection is extremely rare, and has been observed in connection with fat heart, aortic stenosis, chronic nephritis, disease of the central nervous system, and after bodily and mental exertion. The treatment consists in assumption of the *horizontal posture* and the use of *stimulants.*

NERVOUS HEART-PAIN (STENOCARDIA; ANGINA PECTORIS).

Symptoms and Diagnosis.—Stenocardia is characterized by the occurrence of **paroxysmal attacks of pain in the precordium,**

which may radiate for a considerable distance, most frequently to the left arm. The pain often attains such a degree of severity that the patients suffer from a sense of oppression, of fear of death, with a pale and drawn countenance, and cold sweats. Many patients obtain relief by firm pressure of the hand over the heart, or by applying the precordium firmly against a solid body, or by hastening to an open window and obtaining fresh air. The **action of the heart** is usually irregular during the attack of pain. The sounds appear faint, as though masked, and the pulse is noteworthy for its diminished tension. At times **vasomotor disturbances** appear, especially in the form of cold, pallor, cyanosis, formication, and hyperesthesia in the hands and arms. In some cases the attacks of pain are preceded by vasomotor disturbances.

Attacks of the kind described not rarely occur during profound rest, and with especial frequency during the act of going to sleep; or they occur in the sequence of physical and mental exertion, after errors in diet and the like. Their duration varies between a few seconds and several hours. While in many instances a few attacks occur at long intervals, in others they occur several times daily, and they may thus cause life to be a burden. Death is only rarely induced by the disease, and may result from paralysis of the heart, progressive cardiac weakness, or cerebral hemorrhage.

Etiology.—Stenocardia is a rare disease, which usually spares children, and is at all common only *after the fiftieth year of life*. *Men* suffer from stenocardia much more commonly than women. A distinction is made between *symptomatic* and *essential stenocardia*. *Symptomatic stenocardia* is associated with diseases of the circulatory organs, especially arteriosclerosis of the coronary arteries, chronic myocarditis, fat heart, aortic valvular insufficiency, aortic stenosis, aneurysm of the aorta, and pericardial adhesions. *Essential stenocardia* attacks especially pale, nervous, and obese individuals. **Toxic** influences are often operative, especially excessive indulgence in tobacco, tea, or alcohol. At times the affection arises in the course of certain **central neuroses**, such as hysteria, neurasthenia, and hypochondriasis. At other times it has been observed to follow **emotional disturbance** (disappointment in love). Not rarely it is induced by **reflex irritation**, and it may occur as a sequel of gastric, intestinal, hepatic, uterine, and ovarian disease. Sometimes the disorder has been observed to appear in the sequence of **infectious diseases.** In some cases it has developed in association with **mediastinal tumors** and **mediastinitis.** Exposure to cold, also, is considered to be a cause.

The *pathogenesis of stenocardia* has not yet been determined with certainty. The hypothesis that attributes the attacks to transient conditions of ischemia and enfeeblement of the heart-muscle has at present the largest number of adherents. The severe pain is believed to result from involvement of the cardiac plexus, which is constituted of nerve-fibers from the

: and the vagus, and which lies close to the branches of the coro-
s. The intimate relations of this plexus with the left brachial
d explain the pains radiating to the left arm.

osis.—The prognosis depends upon whether the primary
curable or not, and is therefore unfavorable in cases of
.ic stenocardia. Immediate danger to life is, as a rule,
.[1]

nent.—The *treatment of an attack of stenocardia* is ap-
y the same as that of an attack of tachycardia. **Rest, a
oom, fresh air, bits of ice, an ice-bag to the precordium,
ıneous injections of morphin** are most likely to terminate

:en no convincing results from inhalations of *amyl nitrite* and
:rnal use of *nitroglycerin.*

;h consideration should be given to the etiology, for
causal treatment may bring about a cure of the disease.
ıce with the conditions present various remedies will be
uch as nervines, iron, reduction of obesity, and meas-
d against gastric, intestinal, hepatic, uterine disorders,
:.

V. DISEASES OF THE AORTA.

ANEURYSM OF THE AORTA.

ıic Alterations.—Aneurysms of the aorta represent
l dilatations of this vessel. In accordance with their
ated, spindle-shaped, and *cylindric* aneurysms are dis-
ılthough the surface is usually not smooth, but presents
'regularities. Accordingly as the entire or only a
ıe transverse section of the vessel is involved in the
he condition is designated as *axial* or *peripheral.*
vary in *size* between a dilatation of the vessel just
and the circumference of an adult head, and even
most common *seat* of aneurysms of the aorta is the
ɔrtion of the vessel; then follow the arch and the
portion. Usually there is but a single aneurysm,
'eral are known to have been present in the same

aneurysm is opened, its cavity is usually found filled
d recent *thrombi,* which may be so considerable in
the dilatation of the aortic blood-current is neutral-

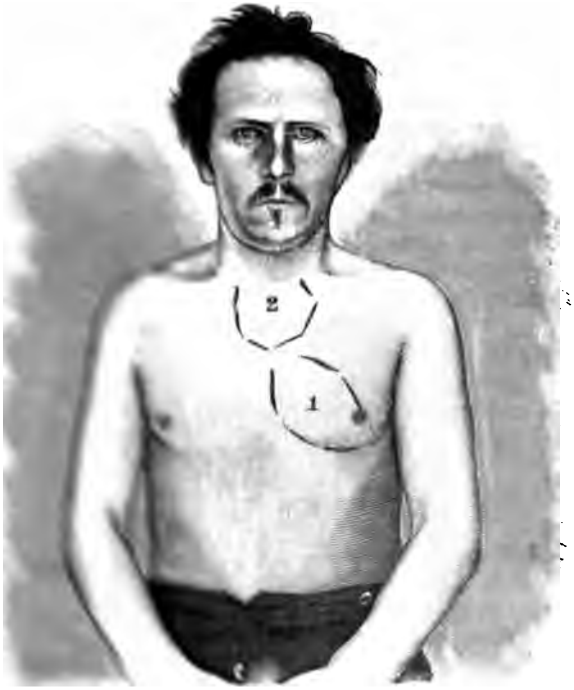
---

an attack of angina pectoris is, however, not rare.—A. A. E.

[illegible] the aneurysm is usually the seat of endarteritic [illegible] fatty degeneration and calcification).

[illegible] examination of the wall of the aneurysm it has been [illegible] elastic layer of the media has disappeared, and some [illegible] considered this the primary cause for the dilatation of the [illegible]

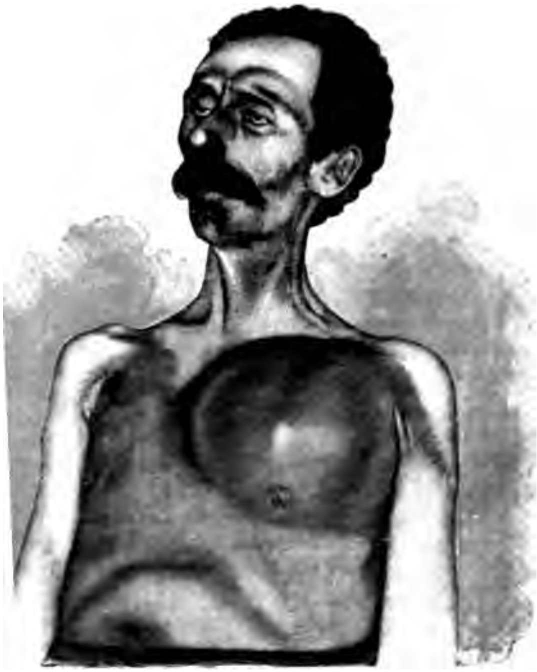
[illegible] As arteriosclerotic alterations predispose to the [illegible] aneurysms, it will not occasion surprise that most



[illegible] aneurysm of the ascending aorta: 1, increased area of [illegible] dulness. (From a photograph (personal observation, Zurich

[illegible] in advanced life, thus beyond the fortieth [illegible] arteriosclerosis and aneurysmal formation early [illegible] be induced by special injurious influences, [illegible] syphilis, alcoholism, gout, obesity, physical over-exertion may be included. *Men* suffer more commonly [illegible] aneurysm of the aorta, by reason of the nature [illegible] In some *countries* aneurysms occur with [illegible] for instance, in England. This probably de- [illegible] upon the extent to which alcoholism and syphilis

ms.—The existence of aneurysm of the aorta is some-
ted only by **suspicious symptoms.** These include ob-
algia in the course of the nerves of the arm or the
nerves, *difficulty in swallowing* in consequence of con-
the esophagus, *epigastric pain,* and *severe vomiting*—all
ns dependent upon pressure exerted by the aneurysm
uous nerves or upon the esophagus or the stomach.
urmurs over the manubrium sterni or in the course
bral column are also noteworthy and suspicious signs.



of the descending aorta, after destruction of the ribs lying immedi-
the skin; from a photograph (personal observation, Zurich clinic).

gnosis may be ventured upon when certain **local**
the circulatory apparatus are demonstrable. The
of these are *abnormal areas of dulness, prominences,*
murmurs. Aneurysm of the ascending aorta and
the aorta are usually attended with *dulness* over
sterni and the adjacent chest-wall, so that a second
is superimposed upon the area of cardiac dulness
urysm of the descending aorta gives rise to dulness
the vertebral column. Naturally, the dulness does
to the entire extent of the aneurysm, but only to

that portion applied to the chest-wall. Not rarely the area of dulness projects above the level of the adjacent surface. The aneurysm may even have caused disappearance of bony portions of the thorax by pressure, and become both palpable and visible as a *pulsating tumor* directly beneath the skin (p. 67, Fig. 10). Protrusion and pulsation may be detected with especial distinctness when viewed with lateral illumination. At times the aneurysm exhibits more violent pulsation than the heart, and an appearance is created of two distinct pulsating bodies functionating in the thorax independently of each other. It may be of importance to introduce the index-finger from the jugular fossa behind the sternum, and to push it downward, when an aneurysm of the ascending aorta or of the arch of the aorta is present, because in this way the dilated and pulsating vessel can be reached with the tip of the finger. *Systolic murmurs* are usually audible over aneurysms of the aorta, for the sudden dilatation of the blood-stream gives rise to blood-whirls within the aneurysm. Murmurs will be wanting only when the aneurysm is so thoroughly filled with thrombi that dilatation of the blood-channel is no longer present.

Not rarely a *systolic* and a *diastolic murmur* are audible over aortic aneurysms. The diastolic murmur is either generated within the aneurysm itself when the diastolic reflux of the blood takes place with such energy that audible blood-whirls occur within the aneurysm, or transmitted from the aortic valve (which has frequently at the same time become incompetent in consequence of atheromatous changes) to the aneurysm. Under the latter [illegible] the [illegible] character of the diastolic murmur over the aortic [illegible]

[illegible] of the ascending aorta and of the arch of the aorta [illegible] displacement of the heart downward and outward [illegible] the left mammillary [illegible] space. Generally [illegible] hypertrophy of the left ventricle [illegible] apex-beat: which [illegible] valvular insufficiency [illegible] In cases of pure [illegible] of the heart [illegible] place in con-[illegible]

[illegible] consideration [illegible] with the seat [illegible] ascending aorta [illegible] with the [illegible] within the [illegible] the pulse [illegible] carotid with [illegible] left radial. [illegible] is situated [illegible] left carotid.

er, the aneurysm is situated between the left carotid and
ubclavian, the right radial pulse and the two carotid
ur synchronously with the apex-beat, while the left
the femoral, on the other hand, appear later. In cases
m of the descending aorta, finally the pulse in the neck
arms is palpable synchronously with the apex-beat,
ccurs later in the femoral arteries.

tion of the pulse can take place in cases of aortic aneurysm
equence of constriction and distortion of the orifices of vessels
to the aneurysm; but under such conditions the retarded pulse
aracterized by deficient fulness.

ve discomfort may be wanting throughout the whole
he disease; but, as a rule, the patients complain of dis-
eating, a sense of constricton and pain in the chest.
palpitation of the heart and **dyspnea** also not rarely

aneurysm has a tendency to increase in size, and this
ource of two dangers, namely, excessive pressure upon
rgans and rupture of the aneurysm. The **pressure-**
vary in accordance with the seat and the direction of
the aneurysm. Aneurysms of the ascending aorta and
tic arch frequently give rise to *unilateral* or *bilateral*
f the recurrent laryngeal nerve. Obstinate and severe
ial neuralgia is also not rarely observed. If motor
r in consequence of the pressure, pareses and paralyses
the arm (principally the left). Aneurysms of the
aorta often give rise to the most distressing *intercostal*
ninal neuralgia. They also frequently cause a *pain-*
f stiffness in the vertebral column, so that patients are
moving about only with difficulty in a position bent
At times a bronchus is compressed, with the develop-
clinical picture of *bronchostenosis* (usually left-sided);
on the esophagus gives rise to *stenosis* of that tube,
nger of death from starvation. Even the *bones* are
esist the advance of an aneurysm. By erosion of
rtion of the chest-wall the aneurysm may come to lie
external integument. In other instances articular
are severed. Even the vertebral column is at times
y an aneurysm, with resulting compression of the
and spinal paralysis; or the aneurysm ruptures and
ts bloody contents into the vertebral canal.

of an aneurysm is favored by excessive physical and
ity, which increase the blood-pressure; but it may
uring complete rest and during sleep. At times rup-
lace *outward* through the skin. Sometimes, after pre-
ration, the skin ruptures at several points, the blood
ling but slowly, so that at the beginning it may be

possible to control the hemorrhage; but at other times a great tear suddenly takes place in the skin, through which the patient bleeds to death within a few seconds. *Rupture into the pericardial cavity* not rarely takes place, the pericardium rapidly filling with blood, and sudden death occurs in consequence of cardiac paralysis. *Rupture into one of the pleural cavities* takes place still more frequently, and with especial frequency into the left pleural cavity. *Rupture into the esophagus*, the *bronchi*, and the *trachea* has also been observed. Less commonly rupture takes place into the *cavities of the heart* or into the *venæ cavæ*. Aneurysms of the descending aorta at times give rise to undermining and detachment of the pleura or the peritoneum through the extravasated blood.

The *duration of an aneurysm* does not, as a rule, extend beyond a year and a half, although observations are on record covering a period of more than twenty years.

The *termination* is usually fatal, death occurring from rupture of the aneurysm or in consequence of pressure-disturbances (stenosis of the esophagus, spinal paralysis), or from progressive marasmus, or at times also from embolism derived from detached thrombi. It should be mentioned that *pulmonary tuberculosis* is not rare in association with aortic aneurysm.

Diagnosis.—The recognition of aneurysm of the aorta is not rarely attended with considerable difficulty, even in cases in which the symptoms are not exclusively suspicious. In the presence of pulsating tumors care should be taken to avoid confusion with solid tumors situated upon the aorta, and receiving transmitted movement from it. Under the latter conditions, however, only elevation and depression take place, but not **expansile pulsation** of the swelling. The location of an aneurysm will be indicated by the situation of the dulness, the tumor and the pulsation that may be present, and the character of the pulse as previously pointed out.

Prognosis.—The cure of an aneurysm through abundant thrombus-formation and obliteration of the sac can scarcely be hoped for, and almost without exception death from rupture or exhaustion, or one of the complications already mentioned, is to be feared within a short time. The prognosis is thus unfavorable.

Treatment.—Individuals suffering from aneurysms of the aorta must observe permanently **bodily** and **mental rest**, and avoid all **irritating articles of diet** (strong coffee, tea, alcohol). A **daily movement of the bowels** should be secured, as expulsive efforts may cause rupture of the aneurysm.

Fasting has properly been abandoned as a means of treatment, because it hastens the loss of strength already taking place.

Rest in bed for months, the constant application of an **ice-bag**, daily applications of **iodoform-collodion**, and the internal administration of **potassium iodid** can be warmly recommended:

loform,	**1.0 (15 grains);**
llodion,	**10.0 (2½ fluidrams).—M.**
topically.	
ution of potassium iodid,	**5.0 : 200 (75 grains : 6½ fluidounces).**
l tablespoonful thrice daily.	

h can be expected from *astringents* (plumbic acetate, tannic acid,
n of ferric chlorid).

treatment has been variously recommended for aortic
especially the introduction of gold needles connected
ositive pole of a galvanic current, so called *electro-*
or the *introduction of foreign bodies* (watch-spring,
ilk thread[1]), in order to induce coagulation within the
ions of solution of ferric chlorid are probably not free
r of embolism.

neous injections of 100 or 200 c.c. of physiologic salt-
taining one or two per cent. of white gelatin have also
ed with advantage.—A. A. E.

of the right subclavian and carotid has been proposed
nent of aneurysm of the arch of the aorta. Among
onspicuous symptoms severe neuralgia especially re-
the frequent employment of morphin subcutaneously.

TION AND OCCLUSION OF THE ISTHMUS AORTA (ISTHMUS AORTAE PERSISTENS).

ction of the arch of the aorta with the descending
the duct of Botal enters into the aorta, is known as
of the aorta. In newborn children a constriction of
f the aorta exists at this point, and which gradually
Under some conditions the constriction may persist,
may even become closed. The distribution of blood
nutrition of the lower half of the body can then be
through the collateral paths that arise from the aorta
sthmus, and anastomose with arteries that arise from
low the isthmus. These *collateral channels*, which
ever seen in healthy persons, become tortuous, pul-
ibrating vessels distended to the size of a finger, and
frequently, also cardiac-systolic murmurs can be
is an important and distinctive diagnostic phenom-
following principal collateral paths may be men-

vian artery, internal mammary artery, anterior inter-
and superior intercostal artery, posterior intercostal
acic aorta.

---

also attended the introduction of long coils of gold wire through
current is passed.—A. A. E.

2. Subclavian artery, transverse artery of the neck, dorsal artery of the scapula, posterior intercostal arteries, thoracic aorta.
3. Subclavian artery, transverse scapular artery, subscapular arteries, posterior intercostal arteries, thoracic aorta.
4. Subclavian artery, internal mammary artery, superior epigastric artery, inferior epigastric artery, iliac artery.

The *character of the femoral pulse* is noteworthy, being greatly retarded in comparison with the apex-beat of the heart, and being further characterized by its slight fulness. The *left ventricle* undergoes dilatation and hypertrophy in consequence of the reduction in the aortic blood-current, and frequently death has been observed to take place from *rupture of the heart* or *cerebral hemorrhage*. In other instances paralysis of the heart takes place gradually, with *weakness of the myocardium* and *general venous stasis*. Patients may, however, attain great age. Nothing is known as to the *causes* of the condition. About one hundred cases have thus far been reported. The *treatment* is confined to the relief of prominent symptoms.

EMBOLISM OF THE AORTA.

Emboli in the aorta are mostly derived from detached thrombi and vegetations on the aortic or mitral valves, less commonly from cardiac thrombi in the left ventricle or from perforating tumors or echinococcus-cysts of the heart. Thrombi in aneurysms or upon arteriosclerotic lesions in the aorta may also become detached and be swept into the aorta as emboli. If the orifice of the aorta be occluded by an embolus, *sudden death* occurs, because the supply of blood to the heart-muscle and to the brain ceases. Frequently emboli reach the point of bifurcation of the aorta, upon which they ride, sending branches into both common iliac arteries. The patients then complain mostly of *sudden pain* in the lower extremity with the lodgment of the embolus. The *femoral pulse* is wanting, and the extremities appear pale and feel cold like those of a corpse. The function of the sensory and motor nerves is abolished, with the development of *anesthesia and motor palsy in the extremities*. If an adequate collateral circulation do not develop promptly, *gangrene of the extremities* takes place, and the patients die in consequence of *septicemia*. From a therapeutic point of view the physician is powerless in the face

PART II.

EASES OF THE RESPIRATORY ORGANS.

I. DISEASES OF THE NOSE.

L CATARRH (CATARRHAL RHINITIS).

gy.—Catarrhal inflammation of the nasal mucous
also designated *coryza*, is a most *widespread disease*.
very person is probably attacked by coryza more than
g his life, it appears that the tendency to suffer from
r is a variable one. Delicate, pale, and slightly built
attacked by coryza with especial frequency and readi-
fulous persons, also, are in marked degree susceptible
rder, and likewise persons who are exposed to the
f dust and irritating gases. Among the causative
tious, *toxic*, and *traumatic* (*mechanical*) *influences* may
mentioned; to these may be added *extension of con-*
mmation.

rhinitis may occur either as an independent and
ease, or as a *secondary* disorder in the sequence of
ous diseases, such as influenza, measles, typhoid fever.
ctious rhinitis is often attributed to **exposure to cold**;
ough it be conceded that coryza may quickly follow
re, this, in all probability, acts only as a contributing
wering the resistance of the tissues and affording
tunity for the invasion and propagation of lower
ganisms capable of exciting inflammation (bacteria)
changes in the circulation in the nasal mucous mem-
s true that nothing positive is yet known as to the
ese bacteria. Bacteriologic examinations have dis-
resence of bacteria in the nasal secretion, but as a
urse nothing is thereby demonstrated, as bacteria can
l in the nasal mucus of healthy persons, being taken
inspired air by the moist mucous membrane. There
ly any doubt that coryza possesses a considerable

areful laymen it is, there
from the disorder.
gents taken internally, thu
ation of irritating gases. I
from the ingestion of potas-
may likewise follow inhala-
acid, nitric acid, chlorin-gas

at times a result of the **inha**
frequently as an *occupation*
have penetrated the nares als
se who indulge in **snuff** ofte
bstance irritating the nasal
ly, in part chemically.
by **extension from adjacent**
nance, in association with ab-

atomic Alterations, and

ce between *acute* and *chronic*
the duration and the course
ases *acute coryza* begins like
ectious disease. The patients
ears, and even for days, as if
the onset of some serious dis-
ment give added justification
lterations in the nose make
prickling sensation becomes
in both. The nares become
and speech acquires a nasal
to frequent sneezing becomes
active secretion takes place
so that in the course of a day
soiled. The nasal secretion
and of a salty taste; after a
ore viscid, and greenish and
number have become added
cellular elements. If the
secretion, slight redness and
the course of a few days the
gin to disappear, and in some
ness sets in.
st followed with the aid of
redness and swelling of the
ses of mucus and pus, and
s can be observed.
rough **extension of the inflam-**
Pain on either side of the

nose, often severe and distressing, is dependent upon
on of the mucous membrane of the frontal sinuses.
isations in the upper jaw indicate involvement of the
Highmore. Marked lacrimation and active redness
ng of the conjunctiva indicate that the inflammation
d the lacrimal duct and the conjunctiva. Catarrh of
membrane of the Eustachian tube (tinnitus aurium,
of hearing), and especially inflammation of the middle
ite unpleasant accidents. Catarrh of the mucous mem-
e pharynx, larynx, trachea, and bronchi is not rarely
to that of the nasal mucous membrane.

catarrh of the nasal mucous membrane frequently de-
repeated attacks of acute nasal catarrh, especially if
akes place before the preceding attack has been cured.
stances, however, *chronic coryza* appears in its chronic
ie outset. The result depends in part upon the causa-
Patients with chronic coryza suffer from impermea-
nares, so that they are compelled to breathe with the
As a result the facial expression acquires a stupid
and the deficient warming of the inspired air predis-
ammatory affections of the upper air-passages. The
ll, and, in connection therewith, the sense of taste
ired. Speech becomes nasal. Usually purulent se-
med in excessive amount, which in part dries upon
membrane as grayish-green crusts and mussel-like
often can be expelled only with great difficulty by
iose. At times the inflamed nasal mucous membrane
bundance of thin fluid, and this condition has been
inorrhea.

copic examination it is found that a distinction must
veen *hypertrophic* and *atrophic chronic nasal catarrh.*
ce of *chronic hypertrophic catarrhal rhinitis* the nasal
brane appears thrown into folds and thickened, so
l passages are constricted. The mucous membrane
rownish-red in color, and widely dilated and tortuous
are often visible upon it. In addition there are pres-
le deposits of mucus and pus and desiccated grayish-
Chronic atrophic catarrhal rhinitis may develop from
hic variety, but appears to occur also as an inde-
ion. The nasal mucous membrane is characterized
attenuation, so that the nasal cavity is noteworthy
roominess. Microscopic examination of the nasal
rane discloses absence of erectile tissue, collection
in the subepithelial layer of the mucous membrane,
ation of the ciliated epithelium into squamous epi-
in part is cornified. Experience has shown that
ic rhinitis develops especially in persons with

broad, depressed nasal bridges, and that in some families it is hereditary.

Chronic coryza may be attended with various *complications*, of which **ozena** is especially annoying. Often this is a sequel of atrophic rhinitis, while in other instances **ulcers** have formed upon the nasal mucous membrane, and have extended from this to the bony substructure, and thus maintain an offensive nasal discharge. It is, therefore, customary to make a distinction between *simple ozena* and *ulcerative ozena*. Not rarely the inflamed nasal mucous membrane undergoes hyperplasia and **nasal polypi** develop. At times **reflex neuroses** arise, and these have been observed with especial frequency in association with hypertrophic catarrh. Among such reflex neuroses bronchial asthma, ocular diseases, psychic disturbances, even deformities of the thorax, scoliosis, and a tendency to abortion have been described, although some of the statements must be received with a certain amount of skepticism. Unpleasant complications may arise from *extension of the chronic inflammatory process* from the nose to contiguous mucous membranes. Among these may be mentioned chronic inflammation of the frontal sinuses, the antra of Highmore, the ear, and the pharynx. Although chronic coryza is a disorder scarcely dangerous to life, it is nevertheless a most obstinate and a trying disease.

Treatment.—Prophylactic measures may be employed in the case of delicate persons and those exposed to the inhalation of dust and irritant gases. In an attack of *acute febrile coryza* recumbency in bed should be recommended, with the ingestion of **hot infusions** (pectoral species, elder-flowers, linden-flowers—a tablespoonful to two cups of hot water). In addition, **inhalations of hot steam** afford great relief. Attempts have been made to abort acute coryza, and for this purpose the following formula of Hagen-Brandt has a certain reputation :

℞ Carbolic acid,	5.0 (75 grains) ;
Alcohol,	15.0 (½ fluidounce) ;
Water of ammonia,	5.0 (75 minims) ;
Distilled water,	16.0 (4 fluidrams).—M.

Keep in a dark bottle with a glass stopper.

Dose : A few drops poured upon three or four sheets of thick bibulous paper, and inhaled every two hours, with the eyes closed, so long as an odor is appreciable.

Severe frontal pain is best relieved by means of salicylic acid, sodium salicylate, or phenacetin (1.0—15 grains—thrice daily). In the treatment of *chronic coryza* various **snuffs** and **irrigations (nasal douche**) have been employed. Astringents and disinfectants have been recommended for the latter purpose. I prescribe irrigation of the nares, morning and evening, with carbolic acid (2.0 : 200) or solution of aluminum acetate (2.0 : 200), and then insufflation into the nose of a small amount of the following powder :

Mercurous chlorid, 3.0 (45 grains);
Alum, 5.0 (75 ").—M.
as a snuff.

reatment of *hypertrophic rhinitis* the galvanocautery
od deal. Crusts can be removed by repeated injec-
ewarm solution of sodium chlorid (1.5 : 200). In the
f *ozena* I have observed the best results from the use
as a snuff, while internally potassium iodid (5.0 : 200
onful thrice daily) may be employed. *Ulcers* should
d with silver nitrate. **Change of residence** not rarely
orable influence in cases of chronic coryza ; for instance,
the sea.

S INFLAMMATION OF THE NASAL MUCOUS EMBRANE (FIBRINOUS RHINITIS).

s inflammation of the nasal mucous membrane is rare.
ed with the formation of fibrinous membranes upon
of the nasal mucous membrane, and which can be
hout loss of substance. Among the *causative factors*
y *chemic and infectious agencies*. The disorder may
t times in consequence of *cauterization* of the nasal
brane. In some instances it is excited by *diphtheria-*
h usually find lodgment upon the nasal mucous
the sequence of pharyngeal diphtheria, and rarely
y, and give rise to nasal diphtheria. It is probable,
t a number of other as yet but little known bacteria
f giving rise to fibrinous inflammation of the nasal
rane. This is true especially of those cases in which
develops in conjunction with other infectious diseases
let fever, typhoid fever, fibrinous pneumonia).

ptoms consist in obstruction of the nares and in
ge, which may acquire a putrid character.

osis is easy when fibrinous membranes are extruded
on rhinoscopic examination.

ment consists in irrigation of the nose with carbolic
) to 4.0 : 100), mercuric chlorid (1.0 : 100), or lime-

-FEVER (AUTUMNAL CATARRH).

.—Hay-fever is known also as *Bostock's catarrh*,
first thoroughly described by Bostock in 1819. It
is a severe catarrh of the nasal mucous membrane,
or as a mixed form. It is attributed to **inhalation
of the Gramineæ**, as pollen-grains have been found
d lacrimal secretions of patients. In support of the
ficance of the pollen of plants is the fact that the

broad, depressed nasal bridges, and that in some fam hereditary.

Chronic coryza may be attended with various com of which **ozena** is especially annoying. Often this is a atrophic rhinitis, while in other instances **ulcers** have fo the nasal mucous membrane, and have extended from bony substructure, and thus maintain an offensive nasal It is, therefore, customary to make a distinction betw *ozena* and *ulcerative ozena*. Not rarely the inflamed n membrane undergoes hyperplasia and **nasal polypi** d times **reflex neuroses** arise, and these have been ob especial frequency in association with hypertrophic cata such reflex neuroses bronchial asthma, ocular disease turbances, even deformities of the thorax, scoliosis, a to abortion have been described, although some of t must be received with a certain amount of skepticism complications may arise from *extension of the chroni process* from the nose to contiguous mucous memb. these may be mentioned chronic inflammation of the the antra of Highmore, the ear, and the phary chronic coryza is a disorder scarcely dangerous to theless a most obstinate and a trying disease.

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Distilled water	16.0	(4

Keep in a dark bottle with a glass stopper.

Dose: A few drops poured upon three or four sh pape and inhaled [illegible] eyes clo [illegible]

[illegible] by me [illegible] grai [illegible] salt [illegible] a [illegible] with [illegible] reason they must change their residence. I have observed good results in the treatment of hay-coryza from the use of a snuff of **calomel** and **alum**:

℞ Calomel,
Alum, each 3.0 (45 grains);
Morphin hydrochlorate, 0.3 (4½ ").—M.
Use thrice daily as a snuff.

Irrigation of the nose with a solution of quinin has also been praised (1.0 : 740). Hay-asthma may be most certainly relieved by means of **narcotics**; for instance, by a subcutaneous injection of morphin.

MYCOSIS NASI.

In isolated cases *molds* (*Aspergillus*), *filamentous fungi* (*Botrytis Bassiana*), and *budding fungi* (*Oïdium albicans, thrush-fungus*) have been found upon the nasal mucous membrane, and yellowish-gray or greenish fungous masses in the nasal discharge. In treatment *irrigation* with carbolic acid (from 1.0 to 5.0 : 200), mercuric chlorid (1.0 : 1000) and *insufflation* of boric acid may be practised.

II. DISEASES OF THE LARYNX.

CATARRH OF THE LARYNX (CATARRHAL LARYNGITIS).

Etiology.—Catarrh of the laryngeal mucous membrane is one of the most widespread diseases. Delicate, anemic, and debilitated individuals are especially predisposed to the affection. The causes are almost the same as those responsible for nasal and bronchial catarrh. A distinction must be made here also between **infectious, chemic, thermic,** and **traumatic** (**mechanical**) causes, to which may be added *laryngeal catarrh by extension from adjacent disease*, and *hypostatic catarrh.*

Infectious catarrh of the larynx may be primary or secondary. Secondary catarrh develops in the sequence of other preceding disease; for instance, whooping-cough, measles, influenza, pulmonary tuberculosis, syphilis. Primary infectious catarrh of the larynx is also variously known as *rheumatic* or *refrigeratory*, as formerly it was attributed to the influence of cold (rheuma). While this influence in the development of laryngeal catarrh cannot be denied, and although this is observed to occur with especial frequency in northern and rugged regions, and in the spring and autumn at the time of variations in the weather, the influence of cold appears to be confined to the circumstance that the lower temperature provides favorable conditions for the invasion of certain bacteria, through altered blood-distribution and diminished powers of resistance, whereas the actual excitants of the disease are bacteria with their toxins. Accurate information as to the nature of these bacteria is, it is true, as yet wanting. In harmony with the infectious nature of the catarrhal conditions of the larynx under consideration is the fact that the disease, like many other infectious diseases, not rarely occurs in epidemic distribution. Infectious laryngeal catarrh must further be included among those infectious diseases that leave a marked tendency to repeated attacks.

Laryngeal catarrh due to chemic influences may result from inhalation of irritating gases (vapors of hydrochloric or nitric acid or of chlorin) or develop after the ingestion of certain substances, among which potassium iodid and potassium bromid are the best known. In the latter event the substances are eliminated from the blood through the laryngeal mucous membrane, in which they excite inflammation. Laryngeal catarrh is a common manifestation in alcoholics. Perhaps chemic influences are operative in the production of the laryngeal catarrh of patients suffering from renal disease.

Among the *thermic causes of laryngeal catarrh* is inhalation of hot vapors.

Laryngeal catarrh due to *traumatic* (*mechanical*) *influences* is frequently an occupation-catarrh, and accordingly of chronic nature. Prolonged speaking, singing, or commanding is a common cause of chronic catarrh of the larynx in teachers, ministers, actors, public speakers, singers, and officers. In other individuals catarrh of the larynx results from the inhalation of dust and smoke (millers, glovers, stone-cutters, smokers).

Hypostatic catarrh of the larynx develops in conjunction with chronic disease of the heart and of the respiratory organs, when the flow of blood from the venæ cavæ to the right auricle is interfered with, because the action of the right ventricle is thereby impaired and all of the blood is not sent into the pulmonary artery with each systole.

Laryngeal catarrh by extension from adjacent disease is at times secondary to similar disease of the nose or of the pharynx above, or of the bronchi and the trachea below.

Symptoms, Anatomic Alterations, and Diagnosis.— The symptoms of catarrh of the larynx require especial consideration, accordingly as the condition is *acute* or *chronic*. *Acute catarrh of the larynx* not rarely pursues the course of a febrile, infectious disease, with initial chill and fever. In other instances elevation of temperature is wanting. In diagnosis the **local alterations** are naturally the most significant. The patients usually complain first of a **sense of tickling** and of **irritation** in the larynx.

is often especially annoying toward evening and during
and thus disturbs sleep. At first a small amount of
sparent mucus is expectorated with difficulty (sputum
Only after some time does the **expectoration** become
lant and looser—that is, more readily ejected—opaque,
sh-yellow (sputum coccum). If the vocal bands are
i the catarrhal process, the **voice** is changed, becom-
impure, and in marked cases completely toneless.
for this resides most frequently in the fact that the
ed thickened vocal bands are no longer capable of
ccurate approximation during speech.

tomic alterations attending acute catarrh of the larynx
t accurately studied with the aid of the laryngoscope,
accurately than on the cadaver, because after death
ons (hyperemia, swelling) may have receded to a con-
gree. The inflamed parts are characterized by exces-
on with blood, and correspondingly appear abnormally
Here and there an isolated tortuous and distended
can be recognized. Besides, the inflamed areas are
he secretion of the mucous membrane is also greatly
id mucous and purulent deposits are often seen upon
of the mucous membrane. Not rarely these have
een the vocal bands, so that long strands of mucus
visible in this situation in phonation. At times the
the mucous membrane with blood is so great that
re and the interior of the larynx is covered with free
iis be removed with a camel's-hair brush, under the
the laryngoscope, it rapidly reaccumulates at times.
ation is at the same time bloody, and the patients are
i great state of alarm by the fear of pulmonary tuber-
ich they consider the expectoration of blood a certain
'his variety of laryngeal catarrh has been designated
aryngitis. At times the epithelial layer of the laryn-
nembrane is exfoliated in places, with the develop-
rhal erosions and ulcers.

cases the inflammatory exudation extends to the
scles, with the development of **paralysis** of individual
st frequently the arytenoid and the internal thyro-
affected (gaping of the posterior third of the chink
s—cartilaginous glottis—in the first, and of the
thirds—ligamentous glottis—in the latter case in

n **signs of laryngeal** stenosis appear suddenly at
sappear within a short time. As the symptoms
e **of laryngeal** diphtheria or of croup, the con-
n **designated** *pseudocroup.* The children often are
ing the night with a cry that they are suffocating.

the lower temperature provides favorable conditions for
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diminished powers of resistance, whereas the actual e
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with insurmountable

asures should not be
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d speaking and singing.
st in bed is to be advised
nfusions may be adminis-
lden-flowers, one table-
a narcotic prescribed for

10.0 (2½ fluidrams);
0.1 (1½ grains).—M.

0.003 ($\frac{1}{20}$ grain);
0.3 (4½ grains).—M.

ch 0.3 (4½ grains).—M.

1.0 (15 grains);
0.02 ([illegible] grain).—M.
e 10 troches.

As long as the inflamed laryngeal mucous membrane secretes viscid mucus **inhalations of alkalies** by means of Siegle's apparatus (sodium chlorid, from 1.0 to 5.0 : 100; sodium bicarbonate, from 1.0 to 5.0 : 100; sodium carbonate, from 1.0 to 5.0 : 100; potassium bromid, from 1.0 to 3.0 : 100) may be recommended at intervals of two hours. When the secretion subsequently becomes more abundant and more diffluent, the alkalies should be replaced by **astringents** (argentic nitrate, from 0.1 to 1.0 : 100; tannic acid, from 1.0 to 3.0 : 100; alum, from 1.0 to 3.0 : 100; solution of aluminum acetate, from 0.3 to 1.0 : 100). In cases of *hemorrhagic laryngitis inhalations of solution of ferric chlorid* (from 0.1 to 0.3 : 100) should be employed. When symptoms of *pseudocroup* arise the fears of the patient should be allayed by gentle encouragement. In addition **derivatives** should be applied to the skin of the neck (alcoholic frictions, sponges dipped in warm water). Should the shortness of breath increase in a noteworthy degree an **emetic** should be administered (solution of copper sulphate, 1.0 : 100—a dessertspoonful every ten minutes until vomiting is induced; or solution of apomorphin hydrochlorate, 0.2 : 10.0—one-quarter of a hypodermic syringeful subcutaneously). Only rarely will it be necessary to resort to **intubation** or **tracheotomy**.

Chronic laryngeal catarrh is usually treated locally by means of **inhalations, applications,** or **insufflations.** In the employment of any of these measures the rule should be observed to change the medicament after the lapse of a few days, as the mucous membrane readily becomes accustomed to the remedy, and then no longer reacts thereto. In practising the procedures named, **astringents** are employed, although it is advisable to use stronger solutions than in the treatment of acute laryngeal catarrh. For insufflation into the larynx tannic acid (0.1—$1\frac{1}{2}$ grains), plumbic acetate (0.05—$\frac{3}{4}$ grain), or alum (0.1—$1\frac{1}{2}$ grains) is employed. At times it is necessary to institute **causative treatment,** and, for instance, in cases of syphilis to employ preparations of iodin and mercury, or in the presence of hypostatic catarrh digitalis and other heart-tonics. Those who can afford it will do well in the presence of obstinate chronic catarrh of the larynx to take a course of treatment during the summer at a **bathing-resort.** The alkaline-chlorin springs (Ems, Selters, Wiesbaden, Gleichenberg), saline baths (Soden, Reichenhall, Baden-Baden, Ischl), sulphur-springs (Neundorf, Weilbach, Stachelberg, Baden near Vienna, Baden in Switzerland, Schinznach, Mehadia, Eaux Bonnes), and earthy springs (Lippspringe, Weissenburg), deserve especial consideration in this connection.

Ulcers of the larynx should be cauterized with the stick of silver nitrate, while *laryngeal polypi* and *papillomata* should be removed by operative measures. In cases of ***chorditis vocalis*** *hypertrophica inferior* an endeavor should be made to cause the disappearance of the hyperplasia by means of potassium iodid (5.0 : 200, a tablespoonful thrice daily), and when the dyspnea attains threatening proportions **intubation** or **tracheotomy** should be practised. **Dilatation of the chink of the glottis** by means of sounds also has been attempted. I have effected a cure in a number of cases of obstinate chronic laryngeal catarrh in the treatment of which all possible remedies had previously been employed without result by means of **inhalations of compressed air** and **faradization of the larynx.**

EDEMA OF THE GLOTTIS.

Anatomic Alterations.—Edema of the glottis consists in infiltration of the submucous tissue of the larynx with transudation or exudation. Accordingly, a distinction is made between *hydropic* and *inflammatory edema of the glottis.* In cases of inflammatory edema of the glottis the fluid poured out into the submucosa may be serous, seropurulent, purulent, and in rare cases even bloody. A circumscribed accumulation of pus is designated an *abscess of the larynx.* The diseased laryngeal structures are conspicuous for their great increase in volume, and if they be punctured fluid usually escapes, when the mucous membrane covering the previously swollen structures becomes wrinkled and collapsed. Further, the swelling has not rarely subsided in the cadaver. In cases of hydropic edema of the glottis the mucous membrane often presents an exceedingly anemic hue, while in cases of inflammatory edema, on the other hand, the mucous membrane may be deeply reddened, although here also a paler hue is present when the accumulated fluid has made the blood-vessels of the mucous membrane empty through pressure. Edema of the glottis is usually best developed upon the epiglottis and the aryepiglottic folds, because in these situations the submucosa is looser and provided with a more abundant network. The structures named are often converted into distorted swellings as thick as a finger, which obstruct the entrance into the larynx. Accordingly, the false vocal bands and the region of the arytenoid cartilages are with especial frequency the seat of edematous swelling.

Etiology.—*Hydropic edema of the glottis* develops with especial frequency in the course of **cachectic diseases** and of conditions of **stasis,** of which nephritis, pulmonary tuberculosis, carcinoma, and chronic diseases of the heart and lungs may be mentioned as examples. At times local causes of stasis are present in the veins of the laryngeal mucous membrane (goiter, enlargement of lymphatic glands, mediastinal tumors, aortic aneurysm), and under such conditions the edema of the glottis may be unilateral and circumscribed. *Inflammatory edema of the glottis* occurs most frequently in association with **disease of the larynx**, as, for instance, after inflammation, ulceration, in association with laryngeal perichondritis, after inhalation of hot vapors, irritating gases, dust, after injuries of the larynx, and after the inspiration of foreign bodies. At times the disorder develops in the sequence of **infectious diseases**, among which typhoid fever, pneumonia, and erysipelas may be mentioned. It occurs at times as a *collateral edema* in cases of parotiditis, angina, and angina Ludovici.

Rarely edema of the glottis is of *angioneurotic origin*, developing in conjunction with angioneurotic edema of the skin and of the pharynx.

Occasionally edema of the glottis has been observed to occur without demonstrable cause—*spontaneous edema of the glottis*, although in some cases the condition may have been erysipelas of the laryngeal mucous membrane. Experience has taught that edema of the glottis occurs more frequently in *men* than in women: and *adults* are more commonly affected than children.

Symptoms.—The manifestations of edema of the glottis consist in the **signs of stenosis of the larynx.** Breathing is visibly embarrassed and *inspiration* especially appears *interfered with* and *prolonged.* Inspiration takes place with a certain degree of caution in order to avoid aspiration of the tumid parts, and thus complete exclusion of air. It is usually stridulous, and occasions *diminution in the total number of respirations.* During each inspiration *the larynx and the trachea are actively drawn downward.* The *nasal alæ dilate* in advance of inspiration, and the *mouth is opened*, gasping for air to a certain degree. The *auxiliary muscles of respiration*, in the neck especially the sternomastoid, contract vigorously during inspiration, while the *intercostal muscles* are depressed, because the lungs are only imperfectly filled with air and distended during inspiration. The *voice* is feeble and not rarely almost toneless. The patients assume an **orthopneic position of the body**, are usually intensely cyanotic, and exhibit an anxious expression of the face. The forehead is covered with cold sweat. The pulse becomes small and frequent, but often retains a fair degree of tension. The skin feels cool. If the dyspnea become excessive, consciousness is gradually lost and death often occurs amid muscular twitchings. In accordance with the *duration and course* a distinction is made between acute, subacute, and chronic edema of the glottis. Acute edema of the glottis persists at times for scarcely an hour, whereas chronic edema may be protracted for days and weeks. In the latter event aggravation and improvement not rarely alternate with each other.

Diagnosis.—While the recognition of stenosis of the larynx is not difficult, difficulties may arise in the way of correctly referring the condition to edema of the glottis. Examination with the

e would naturally lead readily to a correct decision, but
means easy to examine in this way persons suffering
e dyspnea. Not rarely the thickened laryngeal struc-
: felt with the finger introduced through the mouth in
ı of the orifice of the larynx. The history also must
ful consideration.

sis.—The prognosis of edema of the glottis is under
ıs most serious, in view of the danger of death from
which often takes place with surprising rapidity.

ent.—If in a case of edema of the glottis the dysp-
me alarming, relief can be expected only from **operation** (*intubation* or *tracheotomy*), and when demanded
the best instruments at hand must be employed (a pen-
ı the trachea, a hollow pen-holder or a quill instead
cannula). It has further been suggested that the
ucous membrane be scarified with a curved bistoury
h strips of adhesive plaster, except at its free ex-
the fluid be evacuated, or even that the mucous mem-
rated with a sharpened finger-nail, but at the present
gestions are purely theoretic. Under less alarming
empts have been made to dissipate the edema of the
ns of **derivatives** (hot sponges, leeches, counter-irri-
ns to the skin of the neck) and of **emetics** or **purga-**

TION OF THE PERICHONDRIUM OF THE (PERICHONDRITIS OF THE LARYNX).

—Inflammation of the perichondrium of the larynx
ndary disorder, occurring in conjunction with inflam-
eration of various kinds within the interior of the
rhal, tuberculous, syphilitic, carcinomatous, typhoid
ration following smallpox and cholera, may all give
ise. The affection at times occurs as a **primary dis-**
ry of the larynx. The patients are usually *adults*,
ease is uncommon in children.

Alterations.—Perichondritis of the larynx leads
ion of pus between the perichondrium and the car-
rynx. At times the cartilage is completely sur-
, and is exfoliated when rupture of the pus takes
s at times acquires a fetid odor, while the necrotic
s calcified, fibrillated, or cornified. If the abscess-
l cicatrization takes place, deformity and stenosis
ay result. The larynx also at times undergoes
frequently the disease involves the arytenoid car-
commonly the epiglottis.

and Diagnosis.—Perichondritis of the larynx [illegible] recognized with certainty only with the aid of the *laryngo*-[illegible] the seat of the disease being marked by an **intumescence,** which not rarely presents the appearance of pus through the mucous membrane and fluctuates on palpation with the sound. Purulent, [illegible] fetid, expectoration containing fragments of cartilage [illegible] suggestive of perichondritis. **Hoarseness and pain in the** [illegible] **the larynx** are too equivocal as symptoms to be available [illegible] diagnostic purposes. Pain of especial severity usually attends [illegible] of food and fluid when the arytenoid cartilages or the [illegible] the seat of perichondritis. Many patients refuse food [illegible] and prefer to run the risk of starvation. Perichondritis [illegible] may be followed by various consequences. The [illegible] considerable amount of pus may cause such reduc[illegible] cavity of the larynx as to give rise to danger of *death* [illegible] Suffocation may also occur from rupture of the [illegible] pus into the larynx during sleep or from impacted [illegible] fragments of cartilage in the larynx. Death from [illegible] also occur as a result of *edema of the glottis,* which [illegible] added to perichondritis of the larynx. Not rarely [illegible] the larynx gives rise to a febrile state, in which [illegible] **strength** may take place. As may be understood, [illegible] greater when the patient refuses to take food on [illegible] At times the pus from the ruptured abscess [illegible] air-passages, and gives rise to **aspiration-pneu**[illegible] **abscess** or **gangrene.** The pus also at times [illegible] the skin of the neck, where it gives rise to [illegible] erosion of the large vessels of the neck, with [illegible] At times rupture of pus takes place simulta[illegible] larynx and into the esophagus, or into the larynx [illegible] of the neck, with the development of an [illegible] *laryngeal fistula.* Even when the morbid [illegible] *dangers* and *sequelæ* remain. Cicatricial [illegible] of the larynx readily give rise to ***stenosis*** [illegible]

[illegible] prognosis in cases of perichondritis of the [illegible] as the disorder is painful and not unat-[illegible]

[illegible] **treatment** is probably applicable only in [illegible] potassium iodid). Not rarely **surgical** [illegible] incision of abscesses, removal of exfoli[illegible] It is often necessary to relieve pain, [illegible] **applications of cocain** (1.0 : 10) may be espe[illegible] If the expectoration is offensive, **inhalation** [illegible] acid, 2.0 : 100; solution of aluminum [illegible] may be prescribed. Danger of suffocation [illegible] In cases of extensive perichondritis of [illegible] **extirpation of the larynx** has been proposed.

SIS OF THE MUSCLES OF THE LARYNX.

y.—Paralysis of the muscles of the larynx is caused
isease of the related nerve or by changes in the mus-
ture, and accordingly a distinction has been made
urogenous and *myogenous paralysis.* It is not always
decide with certainty as to the origin of a given paral-
ogenous paralysis of the muscles may be of **central,**
r mixed (reflex) origin. Central paralysis occurs espe-
ociation with *disease of the pons Varolii and the medulla*
when the centers for the laryngeal nerves in this
e involved in the morbid process. Such paralysis is
so in cases of acute and chronic bulbar paralysis, mul-
sis, and tabes dorsalis. In cases of ordinary cerebral
also paralysis of the laryngeal muscles upon the para-
frequently present. *Hysteria* is a frequent cause for
the muscles of the larynx. The condition is unat-
anatomic alterations, and constitutes a so-called *func-*
ysis. Paralysis of the muscles of the larynx is often
ith *disease of the vagus,* and especially *pressure-paral-*
origin is not rare. Thus, paralysis of the laryngeal
ot uncommonly observed in conjunction with tumors
(enlarged lymphatic glands, goiter), with mediastinal
rysms of the aorta, pleurisy, pericarditis, carcinoma
agus. Enlarged bronchial glands and contraction of
at times cause paralysis of the recurrent laryngeal
imes the vagus is divided in the neck in the course
removal of goiter, and paralysis of the laryngeal
ts. Whether the *toxic and infectious paralyses of the*
larynx are of central or of peripheral origin is un-
ey have been observed in connection with lead-
aryngeal diphtheria, typhoid fever, and the like.
sis of the muscles of the larynx has been observed to
iation with disease of the tonsils, with the presence
the intestine, and with uterine disease, and to dis-
y after correction of the primary disorder. *Myogen-*
of the muscles of the larynx occurs especially in the
her diseases of the larynx, thus after severe catarrh,
tion with edema of the glottis and perichondritis.
he vocal bands in singing or in speaking may be
aralysis of the laryngeal muscles.

Alterations.—Paralysis of the laryngeal muscles
sted without appreciable anatomic alterations in
les. In other instances degenerative changes are
rves or muscles, or in both.

s.—Paralysis of the muscles of the larynx can be
h certainty only with the aid of the laryngoscope.

A distinction has been made between *respiratory*, *phonatory*, and *mixed paralyses* of the laryngeal muscles accordingly as the paralysis gives rise to disturbances in respiration or of voice-formation, or to both sets of phenomena.

Paralysis of the posterior crico-arytenoid muscles is of the respiratory type, as voice-formation is not at all affected, while inspiration is rendered difficult and stridulous, because the vocal bands do not separate during inspiration, but are brought together at their free margins by aspiration (Fig. 11). The patient is compelled to breathe with great care, as rapid and vigorous respiration readily induces complete closure of the chink of the glottis. Paralysis of the posterior crico-arytenoid muscles takes place not rarely in hysterical patients. If the paralysis is but unilateral, the vocal band upon the paralyzed side remains in the middle line during inspiration (Fig. 12).

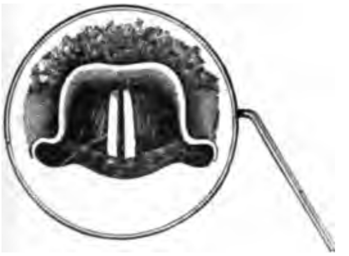

FIG. 11.—Laryngoscopic appearances presented by paralysis of the posterior crico-arytenoid muscles; inspiratory position.

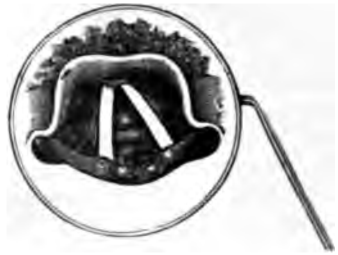

FIG. 12.—Laryngoscopic appearances presented by paralysis of the right posterior crico-arytenoid muscle during inspiration.

Paralysis of the arytenoid muscles causes separation of the posterior third of the chink of the glottis (cartilaginous glottis) during phonation (Fig. 13), while *paralysis of the internal thyro-arytenoid muscles* causes separation of the anterior two-thirds of the chink of the glottis (Fig. 14). Not rarely both pairs of muscles are para-

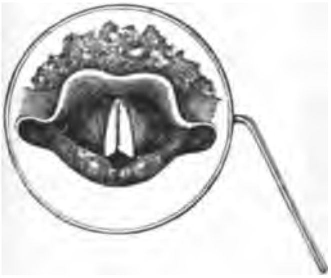

FIG. 13.—Laryngoscopic appearances presented by paralysis of the arytenoid muscles during phonation.

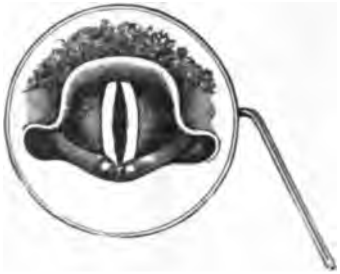

FIG. 14.—Laryngoscopic appearances presented by paralysis of the internal thyro-arytenoid muscles during phonation.

lyzed simultaneously, which may be recognized in the laryngoscope from the fact that during phonation both the posterior third and the anterior two-thirds of the chink of the glottis remain widely open, and are separated by the projecting vocal process of the arytenoid cartilage (Fig. 15). *Paralysis of one internal thryo-arytenoid muscle* causes the vocal band upon the paralyzed side to form a convexity outward during phonation (Fig. 16). *Paralysis of the lateral thyro-arytenoid and the external thyro-arytenoid muscles* cannot be recognized from any peculiarity of the laryngoscopic appearances.

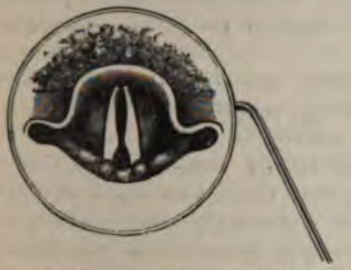

FIG. 15.—Laryngoscopic appearances presented by paralysis of the arytenoid and the internal thyro-ayrtenoid muscles during phonation.

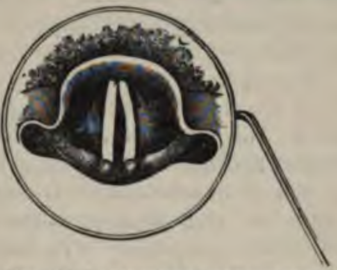

FIG. 16.—Laryngoscopic appearances presented by paralysis of the left internal thyro-arytenoid muscle during phonation.

With *paralysis of one recurrent laryngeal nerve,* which innervates the muscles thus far named, the true vocal band upon the paralyzed side remains immobile during breathing and speaking. It assumes the position that is observed in the cadaver; whence the designation *cadaveric position,* viz., midway between the median position and the extreme lateral position (Fig. 17). In phonation the chink of the glottis is closed, because the healthy vocal band is drawn beyond the median line, and becomes applied to the free margin of its paralyzed fellow. At the same time the arytenoid cartilages overlap, that of the healthy side being placed, as a rule,

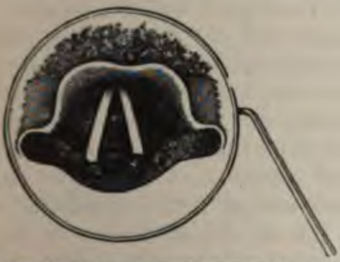

FIG. 17.—Laryngoscopic appearances presented by paralysis of the left recurrent laryngeal nerve; cadaveric position of the left vocal band; inspiratory position.

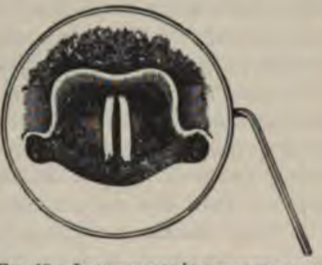

FIG. 18.—Laryngoscopic appearances presented by paralysis of the left recurrent laryngeal nerve during phonation.

(Fig. 18). It is noteworthy that the chin
an oblique course. Changes in the voic
acts of expiration (cough, expulsion) ma

rent laryngeal nerves gives rise to per
the vocal bands, which maintain the sam
ing and speaking. Both vocal bands assum
n. Expulsion, cough, and loud speakin
closure of the chink of the glottis cannot b

ary-epiglottic muscles, which are inner
laryngeal nerve, manifests itself function
aspiration of swallowed substances into th
entrance to the larynx remains open durin
In consequence there is danger of suffo
pneumonia, of pulmonary abscess, and of
On laryngoscopic examination the immo
attract attention.
cricothyroid muscles renders impos-
higher notes, as the tension of the voca
Besides, in the attempt at this, approxi-
cartilage to the cricoid cartilage fails t
laryngoscopic appearances are less distinctive
the middle of the vocal band sinks down-
and projects upward during expiration
of the arytenoid cartilage is not visible
and that in the attempt to produce high
band appears the higher and the longer.
epiglottic and *anterior cricothyroid*
with *anesthesia of the laryngeal mucous*
laryngeal nerve, which innervates the
the sensory nerve for the laryngeal
to the chink of the glottis. In conse-
aspirated particles of food may gain
air-passages, and give rise to inflamma-
gangrene in the lungs, because cough is
capable of causing expulsion from the
foreign body.
are followed by *atrophy from inac-*

prognosis is governed largely by the fact
tions are curable or incurable; but,
of the laryngeal muscles may prove
ment. In cases of hysteria and of
is frequently recurrent.
treatment of paralysis of the laryngeal
first place directed to the **causative**

condition. In addition local treatment should be employed, and by means of insufflations, by the application of sounds, by loud speaking during laryngoscopic examination, by electricity, and by inhalation of compressed air, an endeavor should be made to stimulate the vocal bands into activity. If in consequence of paralysis of the posterior crico-arytenoid muscles danger of suffocation should arise, resort should be had to intubation or tracheotomy. In the presence of anesthesia of the laryngeal mucous membrane, in consequence of paralysis of the superior laryngeal nerve, nourishment through the stomach-tube will be necessary.

SPASM OF THE GLOTTIS.

Symptoms and Diagnosis.—Spasm of the glottis, a common disease of early childhood, results from *spastic contraction of the muscles that close the glottis and of the diaphragm.* Children that have been carried upon the arm, and were quite cheerful, are suddenly seized with irregular breathing. They rotate their eyes, and finally close them; the head falls forward, and breathing ceases entirely. At the same time the color of the face undergoes a change, becoming pale, and then more and more livid. The pulse becomes accelerated and often also irregular, and the veins of the neck become greatly distended with blood. After a few, at most thirty, seconds respiration is again established, with loud whistling or crowing inspiration, consciousness returns, and the children are soon as bright as before the attack. Such attacks may be repeated more than fifty times in the course of a day; in other instances the disease is terminated with a single attack, or the attack is repeated a few times in the course of days or weeks. During the attack the upper limit of liver-dulness can be demonstrated by percussion to occupy an unusually low level, and this has been attributed to spasm of the diaphragm. Not rarely urine and feces are passed involuntarily toward the end of the attack. Spasmodic contraction of the dorsal flexors of the fingers, toes, hands, and feet occurs also, and at times general clonic muscular spasm. Children with spasm of the glottis not rarely present also nervous restlessness, undue susceptibility to fright, disturbed sleep, hyperidrosis, nystagmus, and tetany. The individual attack frequently occurs without obvious cause. In other cases, however, it is induced by psychic disturbances (crying, laughter, fright), by the entrance of foreign bodies into the larynx, or by falling back of the tongue; or it occurs in connection with deranged digestion.

Prognosis.—The prognosis is serious because of the danger of suffocation. This is the more alarming the more frequently the attacks follow one another, the longer they last, and the younger the patient.

Etiology.—As a rule, only *children*, and especially between the sixth and the twenty-fourth month of life, are attacked by spasm of the glottis. Most frequently the children have been nourished artificially, and present signs of *rachitis*. The disease occurs with especial frequency in northern regions, and in the cold months. At times epidemic occurrence has been observed. An especial form of the disorder is known as *spasmus glottidis ablactatorum*, which appears in some children at the time of weaning.

Anatomic Alterations and Nature of the Disease.—Distinctive anatomic conditions sufficient to explain the disease are as yet unknown, and hypotheses have thus been given a free field. The disease has been attributed to irritation of the vago-accessorius or the recurrent laryngeal nerve by enlarged bronchial glands or by an enlarged thymus gland, or by a softened rachitic occiput, and the like. It has recently been maintained that spasm of the glottis is merely a symptom of tetany.

Treatment.—With the onset of an attack of spasm of the glottis all constricting articles of clothing should be removed, and the child should be raised up, as experience has shown that the dorsal decubitus prolongs the duration of the attack, and it should be taken to an open window, or into the fresh air. Respiration should be stimulated by sprinkling cold water upon the chest, by tickling the soles of the feet or the nasal mucous membrane, or by application of ammonia to the nares for inhalation. If respiration is not resumed, the index-finger should be introduced to the entrance of the larynx to determine whether possibly the epiglottis has not been impacted posteriorly in consequence of spasm of the thyro-aryepiglottic muscles. Tracheotomy and intubation, and faradization of the phrenic nerves have also been recommended. If the attacks recur frequently in the course of the day, the little patient may be kept slightly under the influence of chloroform or of ether. For the general nervous condition nervines have been advised, for instance, sodium bromid (5.0 : 100; 10 c.c.—a dessertspoonful—every two hours). In cases of spasmus glottidis ablactatorum the child must again be applied to the breast and the process of weaning deferred for a short time. Among prophylactic measures appropriate nourishment and the treatment of possible rachitis must be considered.

PHONATORY SPASM OF THE GLOTTIS (SPASTIC APHONIA).

By phonatory spasm of the glottis is understood a spastic contraction especially of the tensors of the vocal bands, which sets in whenever the patient makes an attempt to speak. The dis
is thus suggestive of the coördinatory occupati

e unable to speak aloud, and complain principally of a
·essure in the region of the larynx and within the inte-
thorax. The explosive consonants, such as b, p, d, t,
est enunciated. Most frequently the patients are ner-
ysterical individuals, although the affection occurs also
ɔn with tabes dorsalis and multiple sclerosis. At times
cold or over-use of the vocal bands is assigned as the
addition to treatment of the primary condition, care
·, electricity, and applications of cocain (1 : 10) to the
· been recommended.

DISORDERS OF THE LARYNGEAL MUCOUS MEMBRANE.

ıesia of the laryngeal mucous membrane occurs, as has
·ned, in association with paralysis of the superior
rve, and then extends down to the free border of
ıds. At times, however, the anesthesia involves the
down to the mucous membrane of the trachea. The
to be recognized from the fact that irritation of the
cous membrane with a sound induces no reflex move-
ıer, cough is not excited when foreign bodies enter
n swallowing. For this reason such bodies may
ntrance into the deeper air-passages, and give rise to
ımation in the lungs. Hysteria, diphtheria, lead-
l bulbar paralysis are the most common causes of the

ıt should be directed against the primary affection.
ıourishment by means of the stomach-tube and the
f electricity are to be recommended.

thesia and paresthesia of the larynx are attended
f tickling or burning, or even pain, in speaking, so
ts, who are principally nervous and hysterical per-
·ssed by the belief that they are suffering from some
al disease. They speak only in whispers, or avoid
, and endeavor to make their communications solely

ıent should be especially psychic, and should con-
in encouragement and in daily methodical speech-

LARYNGEAL COUGH.

ough occurs principally in pale and nervous per-
ıes assign their trouble to exposure to cold or over-
·. The cough at times appears paroxysmally ; at
ıterrupted, and not rarely acquires a barking or
·ter. At the same time examination of the respi-

ratory organs fails to disclose the slightest alteration. Gentle encouragement and change of residence are the most reliable remedies.

MYCOSIS OF THE LARYNX.

At times *fungous vegetations* occur upon the mucous membrane of the larynx, forming grayish or yellowish deposits. In a case under my observation the fungi consisted of leptothrix-filaments, in another of cocci (staphylococcus pyogenes albus) and bacilli. The fungous masses may be present simultaneously in the pharynx and in the trachea. Cases are also on record in which only the mucous membrane of the trachea was involved. The disorder is exceedingly obstinate. I have observed the best results from inhalations of mercuric chlorid (0.1 : 100).

III. DISEASES OF THE TRACHEA.

The diseases of the trachea usually arise by extension from the larynx or the bronchi, and do not occur as independent affections. If the chink of the glottis be dilated, the tracheal mucous membrane can be observed with the aid of the laryngoscope.

IV. DISEASES OF THE BRONCHI.

BRONCHIAL CATARRH.

Etiology.—Bronchial catarrh is one of the commonest of diseases. It occurs at all periods of life and in both sexes with almost equal frequency; it attacks with especial frequency delicate, anemic, and debilitated persons, whose tissues have in general suffered in their powers of resistance. It is especially prevalent at times when the weather is variable, particularly in the spring and autumn months. Recovery from one attack of bronchial catarrh predisposes strongly to recurrence. A distinction must be made between a *primary* and a *secondary bronchial catarrh* accordingly as the disorder occurs as an independent affection or develops in the course of other diseases. Secondary bronchial catarrh may result by extension of disease from the larynx, the pharynx, the trachea, and the lungs, but it occurs also in conjunction with many infectious diseases; for instance, influenza, whooping-cough, diphtheria, measles, and typhoid fever. The actual injurious agencies may be divided into *infectious, toxic, traumatic* (*mechanical*), and *circulatory.*

In all catarrhal states attributed to exposure to cold (refrigeratory, rheumatic bronchial catarrh) **bacteria** probably play the actual exciting rôle, while the cold merely establishes a favorable medium for their development by causing changes in the distribution of the blood and the powers of resistance of the tissues. The catarrh arising in the sequence of infectious diseases is likewise attributable to bacteria. It is true that but little is as yet known with regard to these organisms. It may be mentioned that influenza-bacilli have been found in the bronchial secretion in cases of influenza-catarrh. The significance of **toxic influences** may be appreciated from the fact that bronchial catarrh develops after inhalation of irritating gases (vapors of acids, chlorin-gas, and the like). The ingestion of certain medicaments (potassium iodid, potassium bromid) also is followed by bronchial catarrh. *Traumatic bronchial catarrh* induced by mechanical irritants occurs especially as an **occupation-catarrh** in persons who by reason of their employment are exposed to the inhalation of dust. Among these may be mentioned millers, bakers, stone-cutters, hat-makers, furriers, waiters, and others. Heavy smokers also, especially when they indulge in the practice of inhaling the smoke, often suffer from bronchial catarrh. As the injurious influences under these conditions are of a persistent character, the resulting bronchial catarrh also is usually chronic in course. Among the varieties of bronchial catarrh induced by **circulatory disturbances** is a *hypostatic catarrh,* which develops especially in connection with diseases of the heart and chronic respiratory diseases, but also with diseases of the abdominal cavity, when the flow of venous blood to the heart is interfered with. Further, in some cases several causes are probably operative simultaneously. If speakers, singers, and those who play on wind-instruments suffer frequently from bronchial catarrh, not only circulatory, but also mechanical influences (alterations in the pressure-relations of the expired air) must be taken into consideration.

Anatomic Alterations.—*Acute bronchial catarrh* is characterized by redness and swelling of the bronchial mucous membrane, which is covered with mucous and mucopurulent secretion. This can be expelled from the bronchi by pressure. In cases of *chronic bronchial catarrh* the mucous membrane exhibits rather a brownish-red or a grayish-red color, and accumulations of secretion are also present. At times **trabecular degeneration** of the bronchial mucous membrane has taken place, as a result of which the mucous membrane, by reason of the marked development of the elastic layer, exhibits prominent and variously branched projections and ridges. At times loss of substance occurs in the bronchial mucous membrane—**bronchial ulcers**; or in other instances papillary excrescences form—**bronchial polypi.** Chronic bronchial catarrh may give rise also to **bronchial dilatation** and hyperplasia of the peribronchial connective tissue, and in this way to **interstitial pneumonia.**

Symptoms and Diagnosis.—In many cases bronchial catarrh is attended only with irritation of the air-passages, cough, and expectoration. Even the most careful examination of the air-passages fails to disclose any local alteration. Such conditions are present when the catarrh is seated at the commencement of the large bronchi. Among the *local symptoms* the occurrence of **rales or rhonchi** is distinctive of bronchial catarrh. **Dry râles,** *rhonchi sicci,* occur when the catarrhal condition is attended with swelling of the bronchial mucous membrane and the production of a viscid secretion—so-called *dry bronchial catarrh.* If the catarrh is seated exclusively in the large bronchi, snoring (*sonorous râles*), *rhonchus sonorus,* occurs, while with catarrh of the finer bronchial tubes whistling (*sibilant râles,*) *rhonchus sibilans,* occurs. Often sonorous and sibilant râles are present together, because the catarrh is distributed throughout the entire bronchial tree. It can then readily be observed that, in accordance with the direction of the current of air, sonorous râles first become audible during inspiration and then sibilant râles, while conversely during expiration sibilant râles are heard first and then sonorous râles. Not rarely the sounds are so loud that they can be heard across a room. They may at times also be felt through the chest-wall as a vibration on palpation—so-called **bronchial fremitus.** In a case of pure bronchial catarrh the percussion-note always remains clear. The respiratory murmur likewise always retains its vesicular character, although it may be unusually sharp, or interrupted or jerky, if the catarrh be irregularly distributed in the smaller bronchial tubes; or the expiratory murmur appears strikingly prolonged in comparison with the inspiratory murmur. **Vocal fremitus** and **bronchophony** at most undergo transitory diminution in cases of bronchial catarrh if the lumen of the bronchi is constricted or temporarily occluded by masses of secretion.

In cases of bronchial catarrh with readily movable or fluid secretion, so-called *moist bronchial catarrh, moist râles,* or *bubbling, rhonchi humidi,* occur. While dry râles are dependent upon stenosis, moist râles are due principally to bursting of the bubbles into which the secretion is thrown by the respiratory air. In accordance with the number of bubbles a distinction is made between abundant and scanty bubbling, and in accordance with their size between large, small, and medium bubbling râles. An abundance of râles is indicative of the fluidity and the amount of the bronchial secretion; while the size, on the other hand, is suggestive of the site of origin, as large-sized râles cannot develop in the smaller bronchi. Most frequently moist râles are heard only during inspiration or during both inspiration and expiration. Rarely they are audible exclusively during expiration. Accordingly as the bubbling takes place in the superficial or the deep bronchi a distinction is made between clear (loud) and dull (faint) moist râles. An intensification of the râles (consonance) does not take place in cases of pure bronchial catarrh. Frequently an attack of bronchial catarrh begins with dry râles and terminates with moist râles, because the bronchial secretion is at first viscid, and only later becomes diffluent.

Cough is a constant and troublesome symptom of bronchial catarrh. It often deprives the patient of rest at night, and not rarely causes severe thoracic pain by overstretching of the muscles. The more closely the catarrh is situated to the bifurcation of the trachea, the more troublesome is the irritation of the air-passages, because the bronchial mucous membrane in this situation is especially sensitive. Severe cough may give rise to vomiting, and also to hemoptysis. It is also frequently attended with distention of the cervical and frontal veins, and as a result of the venous stasis the patients complain of vertigo, mental confusion, and headache. At times severe cough is attended with involuntary evacuation of urine, especially in elderly women; less commonly the bowels are moved involuntarily. At times obstinate cough leads to the development of hernia or prolapse of the rectum.

The **expectoration** varies in accordance with the duration and the nature of the bronchial catarrh, and a variety of forms of chronic bronchial catarrh especially have been distinguished in accordance with the character of the sputum. In cases of acute bronchial catarrh the expectoration is at first scanty, viscid, vitreous, mucous (sputum crudum), and on microscopic examination is found to contain in a mucous ground-substance, coagulating on addition of acetic acid, only scantily distributed mucus-cells or pus-cells (polynuclear round cells). Later the expectoration becomes more abundant and more diffluent, and it exhibits, in addition to the transparent mucous masses, greenish, opaque, purulent points. Such sputum is described as mucopurulent, sputum coccum of a former day.

The **respiration** is accelerated and even embarrassed in cases of extensive bronchial catarrh, so that inspiratory retraction of the intercostal spaces and participation of the auxiliary muscles of respiration may be observed. In cases of chronic bronchial catarrh functional hypertrophy of individual auxiliary muscles of respiration—for instance, the sternomastoid—may gradually take place. The greater the embarrassment of respiration, the more intense does **cyanosis** of the skin and mucous membranes become. **Venous stasis** also develops. The veins of the neck often become swollen to the size of blue cords as thick as a finger, which collapse with each inspiration, to become actively distended during expiration. The lips form thick blue cushions, and the eyeballs protrude markedly on account of the swelling of the retrobulbar veins. Speech appears interrupted and jerky.

In accordance with the *duration* a distinction is made between *acute* and *chronic bronchial catarrh*. *Acute bronchial catarrh* is not rarely attended with febrile movement, though the temperature does not usually attain a high degree, and generally it pursues an irregular course. If the catarrh be seated in the larger bronchi, the patients frequently complain of a feeling of soreness beneath the sternum, in addition to the distressing cough. Catarrh of the smaller bronchial tubes is known also as *bronchiolitis*. In children and aged persons especially this is by no means free from danger, as suffocation may take place and bronchopneumonia (catarrhal pneumonia) may be readily superadded to bronchiolitis. Bronchiolitis is distinguished by the presence of small, widespread mucous râles on auscultation. If the disease pursues an unfavorable course, the cyanosis becomes more and more marked by reason of excessive accumulation of carbon dioxid in the blood, breathing becomes increasingly accelerated and embarrassed, consciousness becomes obscured, and death not rarely takes place in the midst of eclamptic convulsions. The duration of acute bronchial catarrh varies from a few days to four or six weeks.

Chronic bronchial catarrh not rarely persists throughout the whole of life. Naturally periods of improvement and of aggravation occur, the latter usually being dependent upon exposure to cold, and meteorologic conditions, and being attended with fever. A *dry chronic bronchial catarrh* has been described in which patients expel a small amount of tough, vitreous grayish sputum with violent coughing and straining. Conversely, in cases of *simple bronchorrhea* large amounts of mucopurulent sputum are expectorated, the purulent masses at times forming rolled-up, grayish-green opaque clumps—*globular sputum* (*sputum globosum*). *Serous bronchorrhea* likewise is characterized by abundant expectoration, which occurs in the form of a limpid fluid suggesting the appearance of a thin solution of mucilage. *Bronchoblennorrhea* is characterized by copious purulent and readily diffluent sputum. At times the expectoration undergoes decomposition, and a *putrid* or *fetid bronchitis* develops. This same condition occurs with especial readiness when chronic bronchial catarrh has given rise to dilatation of the bronchial tubes and stasis of the bronchial secretion takes place in the dilated bronchial cavities, although putrid bronchitis may occur also in the absence of bronchial dilatation. The sputum under such conditions resembles exactly that of pulmonary gangrene, except that shreds of pulmonary tissue are wanting in cases of putrid bronchitis. The expectoration has a most offensive, penetrating odor, and the air about the patient diffuses a most disagreeable odor, not only in the immediate neighborhood of the mouth, but also at a considerable distance. The patients contaminate the air of the room within a short time, and thus render unbearable the association of others. The patients themselves are also unpleasantly conscious of the disagreeable odor, and not rarely lose their appetite entirely from a feeling of disgust. The amount of sputum is usually considerable (up to 500 c.c. and more per day), and as a rule the sputum is expectorated but seldom by day, though always in large amounts—so-called *expectoration in mouthfuls*. On standing, the sputum forms four layers: an upper frothy layer, a second of grayish masses of pus, then a third usually grayish-green serous layer, and finally, at the bottom, a layer of sediment. The last often contains yellowish or brownish granules—*Dittrich* or *mycotic bronchial plugs*—which when compressed diffuse a peculiarly offensive odor, and on microscopic examination are found to contain innumerable bacteria, fat-drops, needles of fatty acids, and blood-pigment. Some of the bacteria are stained violet or dirty-blue on addition of iodin, but this color appears only in the actual body of the bacteria, while the periphery is stained yellow. These structures have been designated *pulmonary leptothrices*, and the decomposition of the sputum has been attributed to them. At times the putrid sputum loses its disagreeable odor on standing in the air, but this reappears after vigorous shaking.

Chronic bronchial catarrh is a most troublesome disorder, in marked degree restricting the patient in his capacity for work and in the enjoyment of life. The disease is further attended with various dangers. As a result of progressive debility, death may result in aged persons especially, the condition having been formerly designated *phthisis pituitosa*. Much more frequently the dangers reside in the *complications*. Chronic bronchial catarrh is a frequent cause of *alveolar pulmonary emphysema*. When this occurs, the respiratory difficulties are increased, and, in addition, *dilatation and hypertrophy of the right ventricle* may readily occur, because a portion of the pulmonary capillaries are destroyed, and also the circulation in the bronchial vessels is obstructed. If the power of the right heart now fail, *hypostatic manifestations* appear. At first it may be possible to relieve these by means of heart-tonics (digitalis); finally, however, the cardiants fail to exert their effect and the patient dies as a result of excessive stasis. Putrid bronchitis is attended with the danger of *septic complications*, including persistent febrile movement, painful articular swellings, cerebral or spinal abscesses, and the like. The putrefactive process at times also extends to the pulmonary tissue, and gives rise to gangrene and its sequels.

Prognosis.—An attack of acute bronchial catarrh often terminates in recovery within a few days, and becomes dangerous only when the smallest bronchi throughout a vast extent are con-

stricted and occluded. Chronic bronchial catarrh is, to say the least, a troublesome disorder, the cure of which may be anticipated only when it is possible to remove the cause.

Treatment.—*Prophylactic measures* will be required, among others, in such persons as render their body sensitive. It may then be necessary to harden the body intelligently by cold frictions. Those engaged in certain occupations are to be guarded against the inhalation of dust or irritating gases by the proper construction of factories and suitable protective measures. *Causal treatment* is indicated especially in cases of hypostatic catarrh. Obese persons, who likewise exhibit a tendency to hypostatic catarrh, should be recommended to get rid of their superfluous accumulation of adipose tissue by dietetic regulations, or by courses of treatment at Marienbad, Carlsbad, Homburg, or Tarasp. In the *symptomatic treatment* narcotics will be employed when the irritation of the air-passages is unusually severe; for instance:

℞ Bitter-almond water,	10.0 (2½ fluidrams);
Morphin hydrochlorate,	0.1 (1½ grains).—M.

Dose: 10 drops for irritation of the air-passages.

Or,

℞ Powder of ipecacuanha and opium,	
Sugar,	each, 0.3 (4½ grains).—M.

Make 10 such powders.
Dose: 1 powder thrice daily.

Expectorants are generally employed for the removal of the secretion from the air-passages. If the secretion be viscid (sonorous or sibilant râles), the solvent expectorants may be employed (ammonium chlorid, apomorphin hydrochlorate, camphor, potassium iodid); for instance:

℞ Ammonium chlorid,	
Extract of licorice,	each, 5.0 (75 grains);
Distilled water, sufficient to make	200.0 (6½ fluidounces).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours.

For moist bronchial catarrh (mucous râles) the irritating expectorants may be prescribed (ipecacuanha-root, senega-root, anisated solution of ammonia, benzoic acid); for instance:

℞ Infusion of ipecacuanha-root,	0.5 : 180;
Bitter-almond water,	5.0 (75 minims);
Sirup of raspberry,	15.0 (½ fluidounce).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours.

℞ Decoction of senega-root,	10.0 : 180;
Anisated solution of ammonia,	5.0 (75 grains);
Simple sirup,	15.0 (½ fluidounce).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours.

℞ Benzoic acid,	0.3 (4½ grains);
Camphor,	0.05 (¾ grain);
Sugar,	0.3 (4½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

A surprisingly rapid result may be obtained at times from the use of **balsamics**, especially in cases of chronic bronchial catarrh. We recommend especially the use of oil of turpentine, myrtol, or creosote; for instance:

℞ Oil of turpentine, 10.0 (2½ fluidrams).
Dose: 10 drops thrice daily in milk.

Or,

℞ Myrtol, 0.15 (2½ minims).
Make 50 such gelatin capsules.
Dose: 1 or 2 capsules every two hours.

Or,

℞ Creosote, 0.15 (2½ minims).
Make 50 such gelatin capsules.
Dose: 1 capsule every two hours.

Balsamics are to be recommended particularly in cases of putrid bronchitis. Not much can be accomplished by the **inhalation** of medicaments, because these scarcely reach the deeper air-passages. Some patients with chronic bronchial catarrh are much relieved by the employment of **compressed or rarefied air (pneumatotherapy)** by means of the apparatus of Geigel-Mayr or of Waldenburg, or the pneumatic cabinet or bells. A useful remedial measure is frequently a **change of residence.** Persons of means suffering from chronic bronchial catarrh will do well to visit places with a mild, equable climate; for instance, in the autumn and spring Locarno, Montreux, Arco, Gardone, and in the winter Nervi, Bordighera, San Remo, Mentone, Ajaccio in Corsica. In the summer a course of treatment with saline baths (Kissingen, Reichenhall, Soden, Oeynhausen, Ischl), or at alkaline or alkaline-chlorid springs (Ems, Selters, Obersalzbrunn, Rohitsch, Gleichenberg). In cases of bronchial catarrh with the presence of an excessive amount of secretion in the air-passages the administration of an **emetic** is at times indicated; for instance:

℞ Solution of apomorphin hydrochlorate, 0.2 : 10.0.
Dose: From 0.25 to 0.5 (4 to 8 minims) subcutaneously.

Should the emetic fail by reason of the presence of an excessive amount of carbon dioxid in the blood and paralysis of the vomiting center, the patient should be placed in a warm bath and an attempt be made to induce deep inspirations by pouring cold water from a considerable height upon the anterior aspect of the chest, or upon the nape of the neck, thus in the neighborhood of the respiratory center.

FIBRINOUS BRONCHITIS.

Anatomic Alterations.—Fibrinous bronchitis, also designated bronchial croup, is attended with the presence of an exudate rich in fibrin, and in consequence coagulable, which forms tubular casts of the bronchial divisions. These may, as a rule, be readily detached from the bronchial mucous membrane without marked loss of substance, although the epithelium of the mucous membrane has disappeared. Both the mucous membrane and the contiguous external surface of the bronchial cast exhibit frequently small and mostly punctate hemorrhages.

Etiology.—A distinction must be made between *primary* and *secondary bronchial croup* accordingly as the fibrinous bronchitis represents an independent affection or occurs secondarily to preëxisting disease. *Primary bronchial croup* is a most rare disease, and for which often no cause can be elicited. At times the affection has been found to have a familial and hereditary distribution. In all probability bacteria must be looked upon as the true exciting cause of the disease, so that heredity and familial predisposition are responsible only for a tendency on the part of the bronchial mucous membrane to be attacked by fibrinous inflammation. *Secondary bronchial croup* is usually of subordinate importance, and occurs most commonly in conjunction with fibrinous pneumonia. It has been observed besides in association with fibrinous inflammation of the larynx (croup), and at times also with pulmonary tuberculosis, measles, typhoid fever, acute articular rheumatism, and valvular disease of the heart. The occurrence of fibrinous bronchitis has been described also in association with diseases of the skin (herpes, eczema, impetigo).

Symptoms and Diagnosis.—Both secondary and primary bronchial croup can be diagnosed with certainty during life only when fibrinous coagula of the bronchial tubes are expectorated. These usually represent casts of the smaller bronchi in cases of secondary bronchial croup, whereas in cases of primary croup they reproduce at times the ramifications of an entire bronchial tree. In sputum they usually sink by reason of their weight to the bottom of the receptacle in which they are collected, where they generally appear as rolled-up, grayish or yellowish-gray masses, frequently streaked with blood. If they be removed with forceps and be agitated in water, they unfold into a delicate arborization, reproducing with remarkable accuracy the ramifications of the bronchi (Fig. 19).

On transverse section it will be seen that the bronchial coagulum possesses a laminated structure, and that it is at times hollow, at times solid. Not rarely the solid central portion presents a grayish or blackish discoloration due to lung-pigment. At times vesicular enlargements appear on individual branches. On *microscopic examination* fibrin with parallel fibers, isolated round cells, at times crystals in the form of double pyramids (v. Leyden's asthma-crystals), and nests of bacteria are found. The fibrinous coagula are soluble in lime-water and alkalies, while their ground-substance swells in acetic and hydrochloric acids.

The symptoms of fibrinous bronchitis may be exhausted with the expectoration of bronchial coagula. In cases of secondary bronchial croup this is the rule, because usually only a few of the smaller bronchi are involved in the inflammatory process. The conditions are, however, different when the larger bronchi are inflamed, for then **signs of bronchial occlusion** appear, and *suffocation* threatens. The breathing more or less suddenly becomes accelerated and embarrassed. In the distribution of the diseased bronchus occluded by a coagulum, inspiratory retraction takes place. In addition, vocal fremitus and all respiratory murmur are wanting in this area. The threatening manifestations not rarely cease with surprising rapidity, when bronchial coagula of considerable size are expelled and the bronchial passages are again permeable to air. At times bloody expectoration follows, but **hemoptysis** may also precede the formation of the coagula. In one case I observed several days later the expectoration of sputum resembling in appearance the rusty-colored sputum of fibrinous pneumonia, obviously because small bronchial hemorrhages had taken place after the expulsion of the bronchial coagula, and whose blood coloring-matter had undergone disintegration within the bronchi. At times, it is true, fibrinous bronchitis is followed

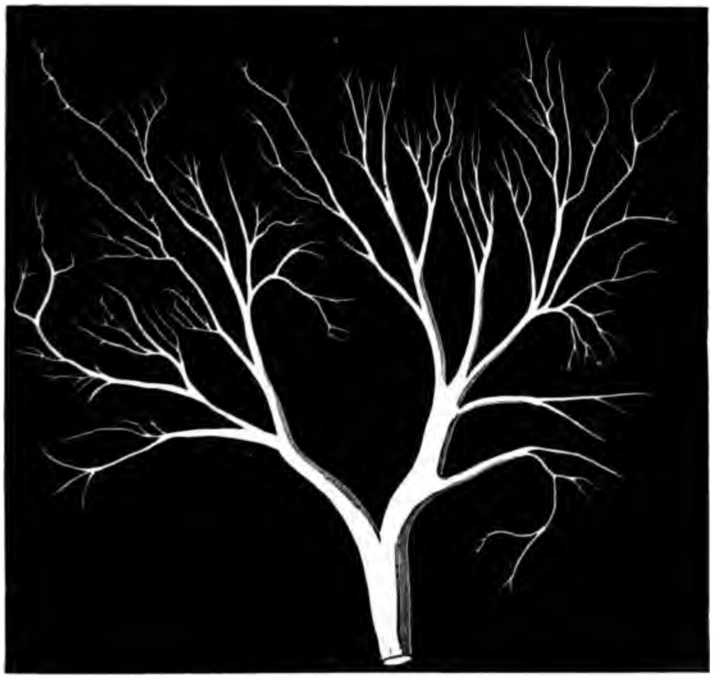

FIG. 19.—Fibrinous bronchial coagulum from a case of primary bronchial croup; natural size (personal observation, Zurich clinic).

by fibrinous pneumonia. Emphysema of the lungs and pulmonary tuberculosis have also been observed in the sequence of fibrinous bronchitis.

The *course of primary fibrinous bronchitis* may be acute, subacute, or chronic, and, accordingly, may extend over a period of a few days, several weeks, or a number of years. The formation of bronchial coagula may recur paroxysmally, and the individual attacks may be attended with chill and fever.

Prognosis.—In cases of secondary bronchial croup the prognosis depends upon the fundamental disorder. In cases of primary fibrinous bronchitis the prognosis is always serious, as each recurrent attack is attended with the danger of suffocation.

Treatment.—In order to avert the dangers of fibrinous bronchitis remedies directed to the *solution of the bronchial coagula* have been employed (lime-water, lactic acid—from 2.0 to 5.0 : 100; potassium carbonate—1.0 : 100; sodium carbonate—1.0 : 100—in the form of inhalations; but probably only a little of the vapor penetrates into the bronchi, quite apart from the possibility of any solvent action). If suffocation threatens, **emetics** (solution of apomorphin hydrochlorate, 0.2 : 100; from 0.5 to 1 c.c.—7½ to 15 minims—subcutaneously) may be administered. For the disorder itself mercurial inunctions and potassium iodid (5.0 : 200; 15 c.c.—1 tablespoonful—thrice daily) have been employed.

BRONCHIAL DILATATION (BRONCHIECTASIS).

Anatomic Alterations.—Dilatation of the bronchial tubes involves most frequently those of medium size, while the smaller and especially the larger are much less commonly affected. The bronchial dilatations are found with especial frequency in the posterior and inferior portions of the lungs, and they are least common in the upper lobe. In accordance with their form a distinction is made between cylindric, spindle-shaped, and saccular bronchiectases. *Cylindric bronchiectases* are characterized by the peculiarity that the bronchial tubes affected do not undergo constriction toward the surface of the lung, but remain at least of equal caliber, and frequently exhibit a bulbous dilatation at the extremity. Such a bronchus has not inappropriately been compared with a glove-finger that has been cut open. *Spindle-shaped bronchiectases* represent gradually increasing and diminishing dilatations of a bronchial tube. If a number follow in close succession, they have been designated as *chaplet-like* or *bead-like*. *Saccular bronchiectases*, finally, depend upon sudden dilatations of the bronchial lumen, and accordingly as the dilatation involves only one lateral wall or the entire lumen of the bronchus a distinction is made between *peripheral* and *axial bronchiectases*. Saccular bronchiectases also follow one another at times in quick succession and thus give rise to a chaplet-like arrangement.

The **bronchial wall** exhibits various alterations in the dilated area. The bronchial mucous membrane at first appears thickened and reddened, but later attenuation takes place, and on microscopic examination it will be found that the cylindric epithelial cells have been transformed into **mucous** or **goblet cells**, and even into **squamous epithelial cells**. Often the mucous membrane presents a lattice-work projection into the bronchial lumen, so-called *trabecular degeneration*, which results from atrophy of the unstriated muscular fibers and the connective tissue, with increased development of the elastic tissue. **Polypoid hyperplasia** of the bronchial mucous membrane, and at times also **bronchial ulcers**, are observed. The **cartilage-cells** frequently exhibit active proliferation, while the cartilage itself disappears.

The **number and the extent of the bronchial dilatations** vary. At times a single bronchiectasis is present, or there may be several dilatations, and in other instances an entire bronchial tree is affected. Not rarely bronchial dilatations are present in both lungs. In the neighborhood of bronchiectasis the pulmonary tissue is usually increased in density and airless, in consequence of interstitial pneumonia, and if a number of bronchiectases are situated close together the lungs acquire the appearance of a coarse-meshed sponge. At times **occlusion** of the outgoing bronchus, but at times also of the ingoing bronchus, takes place. Under such conditions serous or purulent fluid accumulates in the closed bronchial dilatation. This fluid may undergo inspissation, caseation, and calcification, with the formation of **bronchial calculi.** A *tendency toward recovery* is at times exhibited by the formation of granulations upon the inner wall of the bronchiectasis, with adhesions and deformity of its walls.

Etiology.—Bronchial dilatations may be divided into *bronchogenic, pulmogenic,* and *pleurogenic bronchiectases,* in accordance with their etiology. *Bronchogenic bronchiectases* develop most frequently in the sequence of *chronic bronchial catarrh*. They occur also not rarely beyond *bronchial constrictions*, especially beyond occlusion dependent upon aspirated foreign bodies. Further, persistent, severe efforts at cough (as, for instance, in whooping-cough) may give rise to the disorder. *Pulmogenic bronchiectases* are those that develop in the sequence of catarrhal, but more frequently in that of interstitial, pneumonia. Bronchiectasis is also observed in cases of pulmonary tuberculosis. *Pleurogenic bronchiectasis* develops in the sequence of *adhesive pleurisy.*

Uncommon varieties of bronchial dilatation are the *congenital* and the *atelectatic bronchiectases*, the latter of which may be present in atelectatic pulmonary tissue.

From the nature of the causative factors bronchiectasis is more common in *adults* than in children, and it occurs with especial frequency in *men* and in the *lower walks of life.*

The *precise conditions for the development of bronchial dilatations* are not known for all cases, and vary in accordance with the causative factors. Bronchiectasis often results in consequence of exposure of the air within certain bronchial sections to unusually high pressure during inspiration, and especially during expiration. Such conditions are present particularly in cases of bronchial catarrh and stenosis of the bronchi. The development of bronchiectasis is naturally favored when the wall of the bronchial tubes has undergone changes in structure and thus also in its powers of resistance. In cases of interstitial pneumonia the bronchi are not rarely surrounded circularly by newly formed connective tissue, and if this undergo cicatricial contraction it will, by traction outward, cause dilatation of the entering bronchial tubes. If processes of softening have taken place in the neighborhood of bronchial tubes, as, for instance, in cases of pulmonary tuberculosis, the conditions may thereby be provided for the development of bronchiectasis, and the more so as the bronchial wall is frequently also involved and has become more yielding. An unequal distribution of the air in the lungs and over-distention of certain sections of the bronchial tubes with secretion must also be considered as a cause for bronchiectasis.

Symptoms and Diagnosis.—Three symptoms in particular are important for the recognition of bronchial dilatation: in the first place, manifestations of the presence of a **cavity**; secondly, the **expectoration**; and, finally, the **attitude of the patient.**

Signs of the presence of a cavity can only be looked for when the bronchiectasis is situated at the surface of the lung, and possesses a diameter of at least four centimeters. Under such circumstances the percussion-note is tympanitic, or, over cavities as large as an apple, metallic; and at the same time it is usually dull, because the dilated bronchial tubes are prone to be seated in condensed pulmonary tissue. The respiratory murmur is bronchial, and over large bronchial dilatations metallic. Such râles as may be present are ringing (consonating), or over cavities of a considerable size of a metallic ringing character. Vocal fremitus and bronchophony are increased in the area of the cavity. There may also be a cracked-pot sound, Wintrich's, interrupted Wintrich's, Gerhardt's, and respiratory change in note. An important sign of the presence of a cavity consists in the alternation of dulness, absence of respiratory murmur and of vocal fremitus, with a tympanitic percussion-note, bronchial breathing, and ringing râles, accordingly as the cavity is filled with secretion or air.

In the differentiation of a bronchiectatic cavity from a *tuberculous cavity* in the lung it is important to remember that the latter is usually developed in the upper, and the former, on the contrary, in the lower portion of the lung, and that bronchial dilatation is unattended with the presence of tubercle-bacilli in the expectoration, providing that pulmonary tuberculosis has not given rise to the bronchiectasis. Bronchiectasis is to be differentiated from *abscess-formations in the lungs* by the absence of pulmonary tissue from the expectoration in cases of bronchial dilatation. This circumstance is also decisive in the differential diagnosis from *pulmonary anthrax*, for the expectoration may be fetid also in cases of bronchiectasis if it undergoes decomposition, and putrid bronchitis is superadded to bronchial dilatation. It is possible also for confusion to arise in the differentiation between bronchial dilatation and *sacculated pyopneumothorax* that has ruptured into the lung, but the development of both diseases is different, and besides cholesterin-plates and hematoidin-crystals are not rarely present in the expectoration of the last-named disorder and absent in cases of bronchiectasis. Only rarely have blood-crystals been found also in the expectoration in cases of the latter.

Should signs of the presence of a cavity be wanting, the diagnosis of bronchial dilatation will be attended with a good deal of difficulty. It may then be of importance to recognize that *large moist râles* are always present only *in a single circumscribed area*. At times a probable diagnosis can be made from the *appearances of the sputum*. Often, though by no means constantly, the *expectoration is ejected in mouthfuls*, the patient expectorating seldom, but on each occasion large amounts, which at times may be forced from both mouth and nose. The expectoration may be purely purulent and thin, so that it separates into layers on standing. In other instances it presents a *nummular or a globular appearance*. On microscopic examination it will be found to contain no peculiar element. Not rarely numerous fattily degenerated cells, and even needles of fatty acids, are present. Red blood-corpuscles and elastic fibers are present only when loss of tissue has occurred in the wall of the dilated bronchial tubes. In order, under the diagnostically difficult conditions just described, to decide as to the seat of the bronchial dilatation, observation of the *attitude of the patient* is of importance, for, as with all cavities producing an abundant fluid secretion, patients suffering from bronchiectasis also lie upon the diseased side of the chest in order that the cavity may become as completely filled with secretion and occupy as long a time as possible in so doing, and but seldom excite cough. Possibly the Röntgen rays may help to overcome some of the diagnostic difficulties, and at the same time furnish information as to whether an existing bronchiectatic condition is extensive or circumscribed, and single or multiple.

Bronchiectasis is a *chronic disorder*, which may give rise to varied *complications* and *dangers*. Frequently *febrile movement* may suddenly appear for a time, obviously in consequence of absorption of pus from the bronchial dilatation, and characterized by its tendency to a hectic course, by chills and sweats, and its septic character. In addition there may be marked pallor, great emaciation, loss of appetite, and obstinate diarrhea. At times *metastatic inflammation and suppuration* occur in other organs, for instance articular swelling, and cerebral or spinal abscess. Should expectoration be restrained the secretion may undergo decomposition, and symptoms of *putrid bronchitis* appear, as described on pages 100 and 101. At times copious and dangerous *hemoptysis* occurs repeatedly. There is also danger of *amyloid degeneration*, as with all other varieties of protracted suppuration. It is of little significance that at times the terminal phalanges of the fingers and toes undergo bulbous thickening, and the nails become greatly curved, giving rise to the condition that has been designated *drum-stick fingers*. At times bronchiectases rupture into the pleural cavity, and give rise to *pyopneumothorax*. They have also been observed to form hernia-like protrusions through the chest-wall, and to threaten *rupture externally*. At times death takes place after years of suffering amid signs of *asthenia* or in consequence of secondary *pulmonary tuberculosis*. Some patients die amid manifestations of *stasis*, when the contraction of the lung accompanying the bronchiectasis has induced dilatation and hypertrophy of the right ventricle, and subsequently weakness of the dilated heart develops.

Prognosis.—The prognosis of bronchial dilatation is always serious, for no internal remedy capable of curing the condition is known, and the number of possible dangers is by no means small.

Treatment.—Bronchial dilatations can be corrected only by **surgical measures**, exposing them with the knife or the thermo-cautery and draining them, in order to effect closure through the formation of granulations. Nevertheless, complete success can be expected only in the presence of circumscribed bronchiectasis. Hitherto the results of operative intervention have not been overly brilliant, because the diagnosis of circumscribed bronchiectasis was not possible, but perhaps photographic reproduction by means of the Röntgen rays will change this state of affairs. **Internal remedies** are not strictly directed against the bronchial dilatation, but against the contained secretion, and against possible putrid decomposition thereof. The employment of balsamics or of creosote especially is to be recommended; for instance:

℞ Oil of turpentine, 10.0 (2½ fluidrams).
Dose: 10 drops thrice daily in milk.

Or,

℞ Myrtol, 0.15 (2½ minims).
Make 30 such gelatin capsules.
Dose: 1 or 2 capsules every two hours.

Or,

℞ Oil of mountain-pine, 10.0 (2½ fluidrams).
Dose: 10 drops in milk thrice daily.

Or,

℞ Creosote, 0.15 (2½ minims).
Make 30 such gelatin capsules.
Dose: 1 capsule every two hours.

The strength of the patient should be maintained by a nutritious diet.

BRONCHIAL CONSTRICTION (BRONCHOSTENOSIS).

Etiology.—Bronchial constriction may be of *intrabronchial*, *parietal*, or *extrabronchial* origin. *Intrabronchial constriction* includes not only that resulting from aspirated **foreign bodies**, but collections of mucus, pus, blood, fibrinous coagula and parasites that have perforated and become lodged in the bronchi are also well adapted to induce constriction of the bronchial passages. *Parietal bronchostenosis* may be due to **new-growths** and **cicatrices**, and with relative frequency to syphilitic cicatrices. The number of conditions responsible for *extrabronchial* or *compression stenosis* is very large. Among these may be mentioned neoplasms of the lungs, of the pleura, of the mediastinum, of the esophagus, and of the vertebral column, aneurysms of the aorta, retrosternal goiter, and new-growths of the thymus gland.

Anatomic Alterations.—Bronchial stenosis can be readily recognized, as the bronchial cavity at the affected point is constricted and at times occluded. In other respects the anatomic alterations vary in accordance with the individual causative factor. Below the point of constriction dilatation of the bronchial tube not rarely takes place.

Symptoms and Diagnosis.—The symptoms of bronchial constriction manifest themselves in evidences of **embarrassed or suspended respiration** in the distribution of the affected bronchial tube. They are the more conspicuous and the more readily recognizable the larger the tubes involved. If, for instance, a main bronchus is occluded, perhaps by an aspirated foreign body, the corresponding side of the chest will participate but slightly if at all in the *respiratory movements*, and **inspiratory retraction of the intercostal spaces** will become visible. Breathing may be rendered difficult and require the aid of the *auxiliary muscles of respiration*. Inspiration appears especially slowed and embarrassed, and as a result *respiratory action is retarded*. After a time the air in the obstructed pulmonary area becomes absorbed, and accordingly **retraction of the affected side of the chest** takes place, while the opposite side, in consequence of excessive distention of the healthy lung with air, is conspicuous for its prominence. **Vocal fremitus** is diminished or abolished upon the diseased side of the chest because the voice-waves are intercepted at the constricted point in their transmission into the bronchial tube. The *percussion-note* appears more and more deep and tympanitic in correspondence with the increasing absorption of the air in the occluded bronchial area and the progressive reduction in the tension of the pulmonary tissue. No **respiratory murmur** can be heard, and no **bronchophony** or but diminished voice-transmission. Usually, however, **stenotic rales** are audible at some distance, and they may be palpable as **bronchial fremitus**. If bronchostenosis be suddenly removed, as, for instance, through the expulsion of a foreign body, the phenomena described disappear, often with remarkable rapidity. The respiratory disturbances mentioned as attendant upon bronchial stenosis usually give rise to **cyanosis**, which may attain varying grades of intensity in accordance with the situation and the degree of constriction; and, besides, the body often assumes a **passive attitude**, either upon the diseased side of the chest, in order that the healthy lung may engage in respiration as freely as possible, or in the position of orthopnea. Often the dyspnea is manifested in the desperate and anxious *facial expression* of the patient. The forehead is cool and covered with sweat. The action of the **heart** and the **pulse** are accelerated. At times, during inspiration, the pulse becomes small, almost to the point of disappearance, so-called *pulsus inspiratione intermittens s. paradoxus.*

The *development, course*, and *duration* of bronchial stenosis depend upon the etiologic factors. In the case of swallowed foreign bodies the entire clinical picture appears within the shortest possible time, while in the case of new-growths it develops slowly and with gradual augmentation. If the causes are susceptible of removal, the bronchostenosis may last perhaps only a few hours or days, whereas if the changes are slowly progressive it may persist for months. Under the latter condition the interference with respiration becomes greater and greater until death results amid symptoms of suffocation. It is further noteworthy that not rarely *attacks of increased difficulty in breathing* occur suddenly, without noteworthy alteration in the existing conditions being apparent. Accumulations of secretion above the point of constriction may at times be responsible for such conditions, but in other instances these seem to depend rather upon nervous influences. Death may take place in such an attack, although many patients pass through a considerable number of attacks. While the diagnosis of bronchostenosis is usually attended with no great difficulty, the recognition of the causes may at times be impossible. The history and the conditions present in adjacent organs are decisive in this connection.

Prognosis.—The prognosis in a case of bronchostenosis depends upon the *situation* and the *degree of constriction*, and upon whether this is due to removable or irremediable causes. In the latter event it is hopeless.

Treatment.—The first indication of treatment consists in the removal of the cause—**causal therapy.** Various remedies are deserving of consideration in this connection, such as *expectorants, emetics*, and *surgical measures*. If the given indication cannot be appropriately met, **symptomatic treatment** alone remains. Under such conditions resort will have to be had especially to *morphin*, in order to ameliorate the dyspnea and to render the distress at all

BRONCHIAL ASTHMA.

Etiology.—Bronchial asthma is characterized by the occurrence of *attacks of expiratory dyspnea* induced by transitory constriction of the smallest bronchial tubes. This bronchostenosis is sometimes due to spasm of the unstriated muscular fibers that surround the terminations of the bronchial tubes at the point of transformation into the infundibula somewhat in the form of a sphincter; at other times it is dependent upon acute swelling of the bronchial mucous membrane. Under the conditions first named the designation *nervous bronchial asthma* is employed, and the affection is considered a *neurosis of the vagus*, which is believed to innervate the bronchial sphincter. Under the other conditions the affection is designated *catarrhal bronchial asthma.*

Some physicians maintain that in nervous bronchial asthma *spasm of the diaphragm* takes place; but I have hitherto always been able to convince myself of the presence of the respiratory movements of the diaphragm at the time of an asthmatic paroxysm.

Bronchial asthma is an **hereditary** disease in some families. In other instances the patients are individuals in whose families **a nervous predisposition** is inherited. **Acquired nervousness** also furnishes an undeniable tendency to bronchial asthma. Whether irritation of the vagus-center in consequence of hemorrhage, softening, new-growth, or the like, in the medulla oblongata, is capable of giving rise to *central bronchial asthma*, is at least doubtful. Perhaps *toxic bronchial asthma* belongs to the central variety. It has been observed in connection with lead-poisoning, mercurial poisoning, and uremia, and it has been designated saturnine, mercurial, and uremic bronchial asthma. In any event bronchial asthma has been observed in connection with **irritation of the trunk of the vagus** by tumors in the neck and enlarged lymphatic glands. Frequently bronchial asthma arises through **reflex influences**, and diseases of the most diverse organs may excite the affection. Among *diseases of the respiratory organs* themselves may be mentioned morbid turgescibility of the mucous membrane of the nasal turbinates, nasal polypi, chronic rhinitis, polypi of the vocal bands, chronic catarrh of the laryngeal and bronchial mucous membrane. It is noteworthy that some persons are attacked by bronchial asthma after exposure to certain odors, as, for instance, after inhalation of the odor of roses, violets, or heliotrope; and after inhalation of powdered ipecacuanha-root or of chlorin gas. Not rarely the *reflex irritation* arises from the *digestive organs*, and bronchial asthma may be observed in connection with pharyngeal polypi, hypertrophy of the pharyngeal tonsils, chronic pharyngeal catarrh, hypertrophy of the faucial tonsils, constipation, and intestinal worms. At times the ingestion of certain articles of food (for instance, some kinds of cheese) induces bronchial asthma. **Diseases of the genito-urinary apparatus** also give rise to bronchial asthma; as, for instance, displacements of the uterus. Sometimes well-developed bronchial asthma attends **diseases of the heart.**

It has further been contended that *gout* and *diseases of the skin* may be attended with bronchial asthma.

Experience has shown that bronchial asthma attacks *men* more commonly than women. Although it begins at times in childhood, the patients are, as a rule, usually between the twentieth and the fortieth year of life.

Anatomic Alterations.—No anatomic alteration is known to be peculiar to bronchial asthma. Inflammation of the bronchial mucous membrane, distention of the lungs, and pulmonary emphysema are secondary alterations.

Symptoms and Diagnosis.—Bronchial asthma sets in, at times, in the midst of perfect health. The individual attacked may have been healthy and cheerful on retiring to bed at night, and is aroused from profound sleep by an asthmatic paroxysm. In other instances the patient anticipates the advent of an attack from having exposed himself incautiously to some irritant (certain odors, the ingestion of certain articles of food, and the like), which experience has taught is capable of exciting the asthmatic paroxysm. At times the attack may be preceded by nervous manifestations, such as excitability, depression, and general restlessness.

The *asthmatic attack* is readily recognizable. The patient is seized with extreme dyspnea, respiration is labored, deep cyanosis develops, and sonorous and sibilant râles are audible at a considerable distance. The facial expression is anxious, and the face is covered with drops of sweat. The eyes protrude markedly in consequence of stasis in the retrobulbar veins. The blue lips are swollen and the veins of the neck appear bursting with blood. The bodily temperature remains unaltered, although the pulse is accelerated and usually but poorly filled. On careful observation it will be found that in the act of breathing expiration especially is interfered with and is prolonged. This is explicable by the fact that the inspiratory muscles are capable of exerting greater power and of overcoming more readily possible obstructions in the air-passages than the feeble expiratory muscles, as aids to which the abdominal muscles at times act. The number of respirations is, as a rule, diminished. On *examination of the thorax* two phenomena stand out prominently: in the first place, the peculiarly deep tympanitic percussion-note, which has been designated by Biermer as the **box-note,** and also the displacement of the margins of the lungs. Both of these depend upon the fact that the inspired air still enters the pulmonary alveoli with comparative facility, but is unable to escape completely during expiration. As a result overfilling of the lungs with air must take place, and **acute pulmonary distention** thus occurs. Only after termination of the asthmatic paroxysm does gradual escape of air from the lungs take place, so that now the boundaries of the liver, which were displaced downward, and the semilunar space have risen to their usual situation, and the area of cardiac dulness, diminished during the attack, resumes its ordinary extent. The respiratory murmur is not audible, as a rule, during the attack, among other reasons because it is suppressed by the loud sonorous and sibilant râles. The individual attack lasts, as a rule, for several hours, and terminates sometimes rapidly, sometimes gradually. Some patients will be exempt from attacks for months, while in others they may be repeated daily. With the termination of an attack mucopurulent **expectoration** takes place. Often, but by no means regularly, the **asthma-crystals** and **spirals** discovered by v. Leyden can be found. *Asthma-crystals* are pointed double pyramids that often lie in groups in small opaque plugs of sputum. Further, they do not occur in bronchial asthma alone, but they are encountered also in bronchial croup and in the expectoration in cases of echinococcus of the lungs. I have found them also in pleural exudates. They have been found in the blood, the bone-marrow, the juice of the spleen, and in other organs in cases of leukemia.

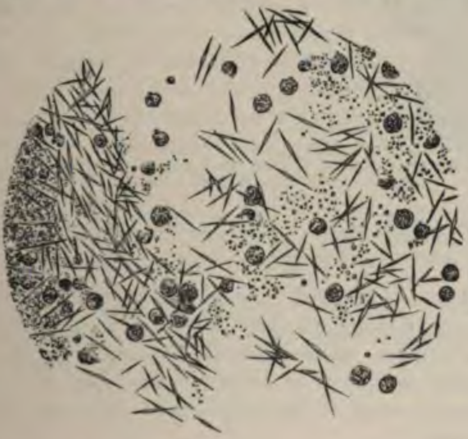

FIG. 20.—Asthma-crystals from the expectoration in a case of bronchial asthma; magnified 275 times (personal observation, Zurich clinic).

In contradistinction from the *sperm-crystals* that resemble them, asthma-crystals are not four-sided, but six-sided, on transverse section.

The *spirals* also have been found not alone in association with

bronchial asthma, but also in cases of fibrinous pneumonia and of bronchial catarrh. They represent coiled, often also fibrillar, filaments, which on microscopic examination not rarely display a bright central thread in their axis, around which at times bright threads yet wind (Fig. 21). In all probability asthma-spirals result from the expression of bronchial secretion through narrow orifices.

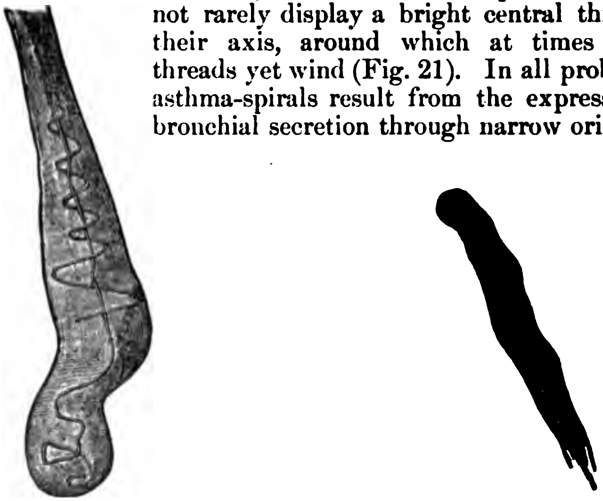

FIG. 21.—Asthma-spirals from the sputum in a case of bronchial asthma; **magnified 275** times (personal observation, Zurich clinic).

Crystals of calcium oxalate (octahedra, envelop-shaped) and of *acid calcium phosphate* are rarely found.

Prognosis.—Although bronchial asthma is a distressing disorder, often persisting throughout life, it scarcely ever causes death, for, when the cyanosis becomes excessive, the spasm of the bronchial muscles relaxes or the acute swelling of the bronchi subsides, in consequence of carbon-dioxid narcosis, and the attack comes to an end.

Treatment.—An *attack of asthma* can be most speedily relieved by subcutaneous injection of morphin:

℞ Morphin hydrochlorate, 0.3 (4½ grains);
Glycerin,
Distilled water, each 5.0 (75 minims).—M.
Dose: from 0.25 to 0.5 (4 to 8 minims) subcutaneously.

However, the asthmatic patient should never be entrusted with a syringe and a solution of morphin, as the danger of morphinism is too great. This danger renders it necessary for the physician to make use of morphin only in the excessively severe attacks, and when these do not occur too frequently. At times, however, the patients have themselves accidentally discovered certain means of amelioration; for instance, the illumination of dark rooms. A large number of remedies have been recommended for the relief of this distressing affection, among which sometimes the one, at other times another, will prove efficacious. Thus, many patients are relieved when paper that has been dipped in a solution of **potassium nitrate** is burned in the room. Others use instead of such paper so-called **asthma-cigarets**, which generally contain stramonium and belladonna. Great stress should be placed in treatment on preventing recurrence of the attacks. This indication is met by **prophylactic** and **causal treatment.** Thus, the patient should carefully avoid such odors and articles of food as experience has shown to be capable of readily inducing an asthmatic attack. Existing diseases of the nose, the pharynx, etc., should be carefully treated. In the presence of chronic bronchial catarrh the employment of compressed or rarefied air should be recommended (apparatus of Waldenburg, Geigel, and Mayer, pneumatic cabinets). Pasty and arthritic patients should undergo courses of treatment at Carlsbad, Marienbad, Tarasp, Vichy, or at similar springs. At times it is an advantage to attack existing nervousness by means of a **strict diet** and an **appropriate mode of life.** In this connection **change of residence** is not rarely of great service. Some patients are free from asthma at mountainous elevations. Among internal remedies **potassium iodid** and **bromids** may be especially recommended. In some cases, also, I have found **arsenic** to be of much good:

℞ Solution of sodium bromid,	15.0 : 200;
Potassium iodid,	5.0 (75 grains).—M.

Dose: 1 tablespoonful thrice daily after eating.

Or,

℞ Solution of potassium arsenite,	
Bitter-almond water,	each 5.0 (75 minims).—M.

Dose: 10 drops thrice daily after eating.

V. DISEASES OF THE LUNGS.

ALVEOLAR EMPHYSEMA OF THE LUNGS.

Anatomic Alterations.—Alveolar emphysema of the lungs gives rise in the first place to dilatation of the alveolar and infundibular spaces. In addition there occurs atrophy of the alveolar septa, so that adjacent alveoli coalesce. Adjacent infundibula also enter into communication with one another, and as a result unusually large air-spaces form in the lungs. As may be understood, destruction of elastic tissue and of pulmonary capillaries is an associated phenomenon. Accordingly, alveolar emphysema of the lungs gives rise to diminution in the elasticity of the lungs, and to increase of blood-pressure in the pulmonary artery.

On *microscopic examination* an accumulation of fatty granules is found around the alveolar epithelium, with atrophy of the elastic fibrous network, and fatty degeneration of the pulmonary capillaries. The last form a loose network, and are remarkably straight, and in places occluded or interrupted.

A distinction can be made between local, unilateral, and bilateral alveolar emphysema of the lungs. The emphysematous portions are conspicuous for their deficiency in blood and in pigment, their light-rose color, an absence of crepitation and a downy consistence on palpation, and the presence of large air-sacs, which at times project beneath the pulmonary pleura as thin transparent bladders up to the size of an apple. The median anterior and the lower margins of the lungs are especially predisposed to emphysematous alterations. In cases of *bilateral alveolar emphysema of the lungs* the **thorax** is often remarkable for its **barrel-like shape.** On opening the chest the **lungs do not collapse.** The lungs cover the heart with their median borders, and upon both sides have pushed the diaphragm markedly downward. Their **margins** appear swollen and rounded, and even with the unaided eye large air-spaces can be made out in them. Usually **chronic bronchial catarrh** exists in addition to alveolar emphysema of the lungs. The heart exhibits **dilatation and hypertrophy of the right ventricle,** and frequently signs of **venous stasis** are present.

Etiology.—Alveolar emphysema of the lungs may be induced by respiratory disturbances of most varied kind, provided these give rise to **permanent increase in the intra-alveolar air-pressure.** Most frequently such disturbances are effective during expiration, less commonly during inspiration. *Alveolar emphysema of the lungs developed during inspiration* is observed in cases of *catarrh of the finer bronchi* (*bronchiolitis*), when, with swelling of the bronchial mucous membrane, the power of the muscles of inspiration is yet sufficient to aspirate air into the pulmonary alveoli, although expiration is too feeble to force the air out again through the constricted bronchial tubes. Exactly these conditions are present *when spasm of the bronchial muscles* takes place, such as has been observed in some cases of bronchial asthma, and in the presence of *foreign bodies in the bronchi.* Under all such circumstances the pulmonary emphysema is preceded by acute distention of the lungs, which may even undergo involution when the causes are removed. *Expiratory alveolar emphysema of the lungs* may develop in connection with all *chronic diseases attended with cough.* For this reason chronic bronchial catarrh is one of the commonest causes of the disorder. In efforts at cough the air in the lungs is exposed to excessive pressure because the thorax and the diaphragm are contracted in expiration, while the chink of the glottis is at the same time closed. The same conditions attend all *expulsive efforts,* and it is, therefore, not surprising that pulmonary emphysema is observed in those who play wind-instruments, in singers, speakers, and such persons as are compelled to carry heavy weights. Mountain-climbers also not rarely suffer from emphysema of the lungs. A special form of alveolar emphysema of the lungs is known as *vicarious* or *compensatory alveolar emphysema of the lungs*, which develops when some portions of the lungs are prevented from taking part in the respiratory process, so that other portions are compelled to assume the functions of the inactive lung. Vicarious alveolar emphysema is not rarely unilateral if the respiratory disturbance involves the opposite lung. Pulmonary emphysema occurs most commonly in *men* and *after the period of puberty*, which is readily comprehensible from the nature of the causative factors.

Symptoms and Diagnosis.—In cases of bilateral alveolar emphysema of the lungs the **shape of the chest** is often conspicuous, the thorax being barrel-shaped, dilated, in a permanent inspiratory position, and emphysematous. The **respiratory movements of the chest** are restricted, and on pressure upon the thorax **increased resistance** is generally found, which is dependent upon *premature ossification of the costal cartilages.* Percussion yields a **deep, slightly tympanitic percussion-note (box-note)**, as the tension of the pulmonary tissue has been diminished. The **expiratory diminution in respiratory pressure** may be demonstrated directly by means of the pneumatometer (the normal expiratory pressure = from 30 to 60 mm. of mercury). The **vital capacity of the lungs** also, which the spirometer shows to be between 2000 and 4000 c.c. in healthy persons, is reduced in cases of pulmonary emphysema. On auscultation the **diminished intensity of vesicular breathing** is striking. The **area of cardiac dulness** is unusually small, or it has completely disappeared, if the median borders of the lungs completely overlie the anterior surface of the pericardium. The **heart-sounds** are remarkably faint, as the lung overlying the anterior aspect of the heart intercepts their transmission. The **upper limit of hepatic dulness** is unusually low (in healthy persons in the right mammillary line between the sixth and seventh ribs) and upon the left the **semilunar space** is much reduced. It is noteworthy, further, that the respiratory displacement of the lower margins of the lungs is exceedingly slight. At times the heart is pushed so far down that not only is its pulsation visible in the epigastrium, but also its right border can be felt. In the presence of unilateral alveolar emphysema of the lungs, the manifestations described are confined to the side of the chest containing the emphysematous structures.

Alveolar emphysema of the lungs is not rarely attended with *complications.* Almost constantly *bronchial catarrh* is present, and it may seriously aggravate the existing respiratory disturbances. At times a subpleural emphysematous vesicle ruptures, and *pneumothorax* develops. Not rarely the vigor of the right ventricle gradually fails, and signs of ***venous stasis*** **appear (cutaneous edema, hypostatic urine, ascites, hypostatic liver, hypostatic catarrh of the stomach and intestines, hydrothorax, hyperemia of the brain, cyanosis, etc.). A large proportion of emphysematous patients die as a result of excessive stasis and of suffocation, after perhaps it had been possible at first to overcome the manifestations of stasis.**

Prognosis.—The prognosis of alveolar emphysema of the lungs is unfavorable, inasmuch as recovery is impossible. On the other hand, however, vicarious pulmonary emphysema must be looked upon as a favorable development. The tendency of emphysematous patients to chronic bronchial catarrh and to myocardial insufficiency is particularly troublesome. Life may, nevertheless, be prolonged for many years.

Treatment.—**Prophylactic measures** should be directed to the avoidance of persistent increase in the inspiratory and expiratory alveolar air-pressure, or the removal of such as may be present. Recovery from or improvement in pulmonary emphysema by means of internal remedies is impossible. At times some relief is obtained through **pneumatotherapy.** To this end the apparatus of Waldenburg or of Geigel and Mayer, or pneumatic bells, may be employed. As in cases of pulmonary emphysema the act of expiration is especially disturbed, the patients should be made to expire into rarefied air. Gerhardt suggested, further, **rhythmic compression of the thorax** during expiration.

INTERSTITIAL EMPHYSEMA OF THE LUNGS.

Etiology.—Interstitial emphysema of the lungs is a rare disease which results from the **escape of air from the pulmonary alveoli into the interalveolar and interlobular connective tissue** of the lungs. Often the air passes in the connective tissue accompanying the bronchi to the hilus of the lung and into the mediastinal connective tissue, whence it finds its way into the subcutaneous connective tissue of the neck, and even beneath the skin of the trunk and of the extremities. The air may also find its way beneath the pulmonary pleura, which it may raise in the form of air-containing vesicles, and these may at times be arranged at the boundary between adjoining lobules like a string of pearls. Should subpleural air-vesicles rupture, pneumothorax will develop.

Among the causes of this disorder are severe efforts at coughing and expulsive efforts, extreme dyspnea, ulcerative disease of the lungs and bronchi, contusions of the thorax and the lungs, too vigorous insufflation of air into the larynx for asphyxia of the

of the skin of the neck (*marked bulging of the skin in the jugular fossa, crepitation on palpation*) or of **pneumothorax,** although all other causes for the two conditions named must first be excluded. Such disturbances as may be present are manifested by **dyspnea.**

As a rule spontaneous absorption of the air takes place after a time, with recovery, so that the **prognosis** is not unfavorable.

If the dyspnea be extreme, relief may be obtained by subcutaneous injection of morphin, but otherwise an expectant attitude should be observed.

ATELECTASIS OF THE LUNGS.

Etiology.—Atelectasis of the lungs indicates **deficiency** or **absence of air in the pulmonary alveoli.** The pulmonary alveoli have either remained airless—*congenital pulmonary atelectasis*—or been deprived of air—*acquired pulmonary atelectasis. Congenital pulmonary atelectasis* occurs in newborn children when the air-passages are obstructed by collections of mucus, aspirated amnial fluid, meconium, or the like, so that distention with air of the pulmonary alveoli that have been rendered airless during uterine life is impossible; or when the respiratory muscles are not sufficiently powerful to dilate the thorax adequately; or, finally, when the irritability of the respiratory center in the medulla oblongata is so greatly diminished that a sense of the need of air does not arise.

Acquired pulmonary atelectasis may be either **obstructive, compressive,** or **marantic.** If *bronchi are obstructed* by mucus, blood, fibrinous exudation, or foreign bodies, or by pressure from without, the air in the alveolar area cut off from the air-supply will be absorbed by the blood and pulmonary atelectasis will develop. *Compression-atelectasis* develops most frequently in association with pleural affections (pleurisy, pneumothorax, pleural tumors) that give rise to compression of the lungs to the point of airlessness. It occurs also in association with pericarditis, dilatation and hypertrophy of the heart, aneurysms, mediastinal tumors, and deformity of the spinal column, as well as with abdominal tumors, meteorism, ascites, peritonitis, and tumors of the lungs. *Marantic pulmonary atelectasis* develops in association with severe disease, such as typhoid fever, as the patient maintains the same posture, so that the respiratory activity of some portions of the chest is in consequence interfered with. In addition weakness of the respiratory muscles is often a contributory factor.

Anatomic Alterations.—Atelectatic areas are most common upon the surface of the lungs. They are conspicuous by appearing depressed and by not crepitating upon pressure with the finger, and they present a dark-red or grayish-red color. On section no air-bubbles escape from them on pressure. Atelectatic portions of lungs sink in water. In contradistinction from inflammatory processes in the lungs they can be readily again filled with air from the supplying bronchus. When atelectasis of the lungs has existed for some time, **inflammatory alterations** may take place in the pulmonary alveoli, and it will then no longer be possible to fill the airless portions with air artificially.

Symptoms and Diagnosis.—As may be understood, extensive atelectasis of the lungs causes **disturbances in respiration.** The patients breathe rapidly and with difficulty, are cyanotic, and become stupid. Physical examination of the lungs discloses signs of **airlessness of the pulmonary tissue** (increased vocal fremitus, dulness on percussion, bronchial breathing); but it is distinctive of pulmonary atelectasis that these phenomena disappear within a short time when the position of the patient is changed, and he is urged to fill the atelectatic portions of the lung with air again. Small atelectatic areas remain concealed during life, or at best they may be recognized on auscultation by the fact that deep inspiration is attended with transitory crepitating râles, when the pulmonary alveoli are again filled with air, and their walls are separated from one another. All of the alterations are best developed in the posterior inferior portions of the lung.

Prognosis.—Pulmonary atelectasis is a serious disease, as in debilitated persons it can scarcely be avoided, and frequently it cannot be successfully treated.

Treatment.—In the presence of febrile and debilitating disease **prophylactic treatment** will be required. Measures should be directed to the relief of the debility and the fever, and care should be taken to have the patient change his posture frequently. The latter is also important in the presence of persistent atelectasis. Baths, with cold douches, which stimulate deep inspirations may be recommended. In addition efforts should be made to remove the cause of the disorder.

HYPOSTASIS OF THE LUNGS.

Anatomic Alterations.—Pulmonary hypostasis develops when patients have persistently occupied the same posture, and at the same time the power of the heart has failed. Under such conditions the movement of blood is checked in the pulmonary capillaries and veins in portions of the lungs the most dependent and restrained in their participation in the respiratory movements, principally in the posterior and inferior sections. The affected portions present an intensely dark-red color, and on pressure give exit to viscid, bloody fluid. Usually the diseased tissue is deficient in air. After a time blood-plasma may accumulate in the pulmonary alveoli, and it may be admixed with desquamated alveolar epithelium and with colorless and red blood-corpuscles.

Hypostatic pneumonia is often spoken of under such conditions, although inflammatory changes are generally wanting.

Etiology.—The most common cause for pulmonary hypostasis consists in **febrile infectious diseases,** particularly typhoid fever. The condition develops also in the **paralyzed,** in persons suffering from **diseases of the bones and joints,** who are compelled to maintain the same posture. Not rarely it occurs during a **prolonged death-agony.** Conditions that interfere with the expansion of the lungs (ascites, meteorism, abdominal tumors) favor the development of pulmonary hypostasis.

Symptoms and Diagnosis.—Pulmonary hypostasis, if at all extensive, gives rise to **respiratory disturbances** (accelerated breathing, cyanosis, increasing mental confusion). As long as the pulmonary alveoli still contain air the percussion-note will be tympanitic, and the vesicular murmur enfeebled in the area of altered pulmonary tissue. If, however, the pulmonary alveoli are occupied by fluid, there may be increased vocal fremitus, dulness, bronchial breathing, and crepitant râles. The expectoration is frequently conspicuous for its bloody appearance. Pulmonary hypostasis is distinguished from inflammatory conditions of the lungs by the absence of febrile manifestations. In the presence of pulmonary edema râles are audible over an extensive area.

Prognosis and Treatment.—The prognosis is usually unfavorable on account of the serious nature of the causative conditions. Prophylactically much can be accomplished in cases of severe disease by **frequent change of posture** and the administration of **cardiants** for the heart (digitalis, stimulants, baths). The same measures should also be directed against the existing hypostasis of the lungs.

EDEMA OF THE LUNGS.

Anatomic Alterations.—Pulmonary edema is characterized by the presence of **serous fluid in the pulmonary alveoli,** although the interstitial connective tissue is also infiltrated with the same fluid. In accordance with the distribution of the morbid process a distinction is made between *circumscribed* and *generalized edema of the lungs.* On opening the thorax edematous lungs do not collapse, do not crepitate on palpation, but feel soft and down-like, and on section permit the expression of an abundance of frothy fluid, which at times is clear like water or from yellowish to reddish in color, and at other times, in the presence of considerable carbon-particles in the lungs, is dark, or when brown induration of the lungs is present is brownish, and when jaundice exists is icteric.

Etiology.—*Several varieties of pulmonary edema* must be **recognized.** *Hypostatic edema of the lungs* occurs when the left **ventricle fails in power,** while the right continues to act with undiminished vigor. The pulmonary capillaries then become unduly filled with blood. The same condition frequently arises as an **agonal edema** during the death-struggle. Similar conditions are, however, also observed not rarely in association with diseases of the heart and the respiratory apparatus. At times pulmonary edema is dependent upon **abnormal permeability of the blood-vessels.** This may be present, for instance, in nephritis, carcinoma, tuberculosis, and debilitating diseases generally. In rare instances **disturbances in the innervation of the pulmonary vessels** appear to give rise to pulmonary edema, which has thus been observed in association with angioneurotic edema of the skin. **Abnormally low pressure** exerted upon the pulmonary alveoli may also be a cause of pulmonary edema. Such a condition has been observed in the sequence of puncture for pleural effusion, in association with occlusion of the bronchial tubes by foreign bodies or exudation, and after inhalation of irrespirable gases. Considerable importance is further to be attached to *inflammatory edema of the lungs.* This occurs at times at the periphery of inflammatory areas in the lung, at other times as an independent affection, and it is then perhaps appropriately designated *serous pneumonia.* The drinking of cold liquids, exposure to cold in general, and hot baths have been observed as provocative of such conditions.

Symptoms and Diagnosis.—The presence of fluid in the pulmonary alveoli is indicated by **crepitant (fine mucous) rales,** which are generated during inspiration by the separation of the walls of the dilating pulmonary alveoli from the alveolar contents. As fluid is present in the bronchial tubes as well as in the alveoli, it is not surprising that medium-sized and even large mucous râles are audible in addition to small mucous râles. In the presence of extensive pulmonary edema innumerable râles are audible at all parts of the chest. In addition a **tympanitic percussion-note is not** rarely elicitable, because the tension of the pulmonary tissue is diminished. If the fluid in the pulmonary alveoli is unaccompanied by air, dulness, bronchial breathing, and increased vocal fremitus will be present. A distinctive feature of pulmonary edema is further the **expectoration,** which is characterized by its abundance, fluidity, and frothy appearance—*serous expectoration*—so that it has been compared with white of egg that has been beaten into snow and subsequently liquefied, and with soapsuds. Its color is usually light yellowish or reddish, from admixture with blood. If pulmonary edema be superadded to fibrinous pneumonia, the expectoration acquires the appearance of prune-juice. The presence of considerable quantities of fluid in the air-passages frequently gives rise to loud bubbling râles over the chest, and the phenomenon has also been designated as **boiling in the chest.** If expectoration be interfered with and fluid accumulate in the trachea, the **tracheal rales** so much and justly feared develop, and which so frequently appear in the death-struggle. The patient suffering from pulmonary edema exhibits signs of **objective dyspnea,** which are manifested in accelerated breathing, inspiratory retraction of the intercostal spaces, participation of the auxiliary muscles of respiration, cyanosis, and, in consequence of increasing accumulation of carbon dioxid in the blood, in progressive stupor. Pulmonary edema may cause death within a short time from *suffocation.* In other instances it persists for days, or appears and disappears repeatedly, so that a distinction must be made between *acute*, *chronic*, and *recurrent pulmonary edema.*

Prognosis.—The prognosis of pulmonary edema is serious on all occasions, as the condition is often dependent upon incurable disorders; and besides, whenever the edema is extensive, there is danger of death from suffocation.

Treatment.—In cases of pulmonary edema **venesection** is indicated as an important measure should the pulse be strong.

Bandaging of the extremities (by means of rubber tubing or compresses), in order to diminish the flow of venous blood to the right heart, is a less reliable procedure.

In addition, **stimulants** and **expectorants** should be employed to increase the force of the heart and to clear the air-passages through increased expectoration; for instance:

℞ Powdered digitalis-leaves,	0.1	(1½ grains);
Benzoic acid,	0.3	(4½ grains);
Camphor,	0.05	(¾ grain);
Sugar,	0.5	(7½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

The administration of strong wine can be warmly recommended. If the patients are unable to swallow, **camphorated** oil may be injected subcutaneously (15 minims from every two to four hours), and dry cups and mustard-plasters or alcoholic frictions to the chest should be prescribed.

CATARRHAL INFLAMMATION OF THE LUNGS (CATARRHAL PNEUMONIA).

Etiology.—Catarrhal pneumonia is always a *secondary disorder,* developing in the sequence of preceding inflammation of the smaller bronchi—bronchiolitis. The disease is, therefore, also designated *bronchopneumonia.* In some instances the inflammatory process extends directly from the bronchial mucous membrane to the pulmonary alveoli, while in others exciting agents of inflammation are aspirated from the bronchial tubes into the pulmonary alveoli, where they give rise to secondary inflammation. Bacteria are looked upon as the *exciting agents of inflammation,* although various kinds must be taken into consideration. Fränkel's *pneumonia-*

cocci, which act also as the causative agents of fibrinous pneumonia, are found with especial frequency. The Streptococcus pyogenes and the Staphylococcus pyogenes aureus and albus are next in frequency as exciting factors. Friedländer's *pneumococci* have also been described as causative agents. The bacteria named are frequently found in the inflammatory foci in pure culture. Occasionally other specific bacteria are found accidentally; as, for instance, diphtheria-bacilli. *Age* has an important influence upon the development of catarrhal pneumonia, for the disease, as a rule, attacks *children* or the *aged*. Diminished powers of resistance on the part of the pulmonary tissue, smallness of caliber of the bronchial tubes in children, and deficient activity of respiratory movement in the aged may be responsible factors. Delicate, anemic, rachitic, and scrofulous children are attacked with especial frequency, as well as those compelled to live in over-crowded, dusty rooms with an insufficiency of light and of air. Catarrhal pneumonia occurs with especial frequency in connection with infectious diseases, particularly in children with measles, whooping-cough, and diphtheria. It occurs not seldom in the form of a foreign-body pneumonia or an aspiration-pneumonia, when saliva, food, or fluid gains entrance into the air-passages and reaches the alveoli of the lung. Similar conditions are observed in those profoundly ill (typhoid fever), in the debilitated (carcinoma, pulmonary tuberculosis), in those suffering from paralysis of the muscles of deglutition (bulbar paralysis, pharyngeal diphtheria), and in connection with frequent vomiting and regurgitation (carcinoma of the esophagus). Inhalation of irritating gases must also be considered a cause of catarrhal pneumonia.

Anatomic Alterations.—Areas of catarrhal pneumonia are almost always situated at the surface of the lung, where they are conspicuous for their dark-red color and dense, airless consistency. On section no air can be expressed, but in contradistinction from atelectatic areas, which are not rarely found side by side, the pneumonic areas cannot be filled with air from the bronchus, and when placed in water they sink to the bottom. Usually numerous small foci of inflammation are found, which exhibit a lobular arrangement; hence the name lobular or insular pneumonia. These are usually especially numerous in the posterior and inferior portions of the lungs. At times they coalesce and involve the larger portion of a pulmonary lobe. With comparative frequency they form airless bands, which begin at the side of the vertebral column at the base of the lung and extend upward, growing gradually smaller and smaller. Generally the inflammatory areas are situated in both lungs. The lung on section is usually smooth, although granular areas of inflammation occur which to a certain degree represent a transition to fibrinous pneumonia. Such a transition may be observed also microscopically. Although the majority of the pulmonary alveoli are filled with a fluid exudate consisting of blood-plasma, desquamated and swollen alveolar epithelial and round cells, fibrinous coagula can be detected in others. In addition to **atelectasis, vicarious pulmonary emphysema** is not rarely found in association with catarrhal pneumonia. **Hyperemia, hemorrhage, and fibrinous deposits upon the pleura** are also common. The **bronchial glands** are usually enlarged and hyperemic.

Symptoms and Diagnosis.—It is often extremely difficult to detect the presence of areas of catarrhal inflammation in the lungs, because, by reason of their small size, they are not accessible to the methods of physical examination and are often concealed behind the manifestations of bronchial catarrh. Suspicion of bronchopneumonia should always be aroused when, in addition to signs of bronchial catarrh, high fever (above 39° C.—102.2° F.) persists for a number of days. In the diagnosis auscultation is of especial value, frequently disclosing the presence of **bronchial breathing** and **ringing rales** over even small bronchopneumonic areas. Dulness on percussion can be expected only when inflammatory areas have coalesced and attain a circumference of five centimeters and a thickness of two centimeters. Even then light percussion is necessary. The impaired percussion-note is likely to be at the same time tympanitic, as the tension of the pulmonary tissue is diminished. Usually **signs of disturbed interchange of gases in the lungs** are conspicuous, as manifested by accelerated and frequently gasping and moaning respiration, by inspiratory retraction of the intercostal spaces, participation of the auxiliary muscles of respiration, and cyanosis. Often there is distressing **cough**; but both children and the aged are given to swallowing the **sputum,** so that this is rarely submitted for inspection. It is then found to be mucopurulent and free from peculiarity. On coughing and on deep inspiration **pain in the chest** is frequent in consequence of over-stretching of the thoracic muscles. The *course of catarrhal pneumonia* may be acute, subacute, or chronic, and extend over a period of from two to eight weeks and more. Not rarely improvement and aggravation alternate with each other. Death results as a rule in consequence of asthenia or of suffocation. Not rarely progressive stupor develops toward the close of life in consequence of excessive accumulation of carbon dioxid in the blood (carbon-dioxid narcosis). There may also be twitchings in some muscles and Cheyne-Stokes breathing. Should the disease terminate in recovery, this takes place but slowly; a crisis does not occur. Further, the symptoms of bronchial catarrh frequently persist for a long time after those of the pneumonia have subsided. Among *complications* pleurisy, pericarditis, endocarditis, and nephritis may be mentioned. At times miliary tuberculosis develops and leads to a fatal termination.

Prognosis.—Catarrhal pneumonia is always a serious affection. In some epidemics of measles and whooping-cough the largest proportion of children attacked by bronchopneumonia die. The more extensive the inflammatory process, and the higher the fever, the greater is the danger to life.

Treatment.—Individuals with catarrhal pneumonia should be placed in a bright and airy **room**, which can be ventilated directly in the summer, and in the winter through an adjacent room. The **air** of the room should be kept moist by placing a solution of sodium chlorid (0.7 per cent.) or of carbolic acid (2 per cent.), or creosote or oil of turpentine (15 drops on the surface of water), in a vessel upon a stove during the winter, or by diffusing one of these three or four times a day by means of a suitable spray-apparatus during the summer. In addition a bath at a temperature of from 26° to 28° R. (90.5° to 95° F.) and of from fifteen to thirty minutes' duration should be given at nine o'clock in the morning and at four o'clock in the afternoon. If the fever be persistently high, an effort should be made to reduce it by means of a **cold pack** or **phenacetin** (from 0.3 to 0.5—4½ to 7½ grains). **Expectorants** should be prescribed to remove the secretion from the air-passages; for instance:

℞ Infusion of ipecacuanha-root, 0.3 : 100.0 (4½ grains to 3¼ fluidounces).
Simple sirup, 20.0 (5 fluidrams).—M.
Dose: 5 c.c. (1 teaspoonful) every two hours.

Should, nevertheless, excessive accumulation of carbon dioxid in the blood or of secretion in the bronchial tubes take place, a warm bath should be prescribed, while cold water is poured from a considerable height upon the chest. The resulting deep respiratory movements will then help to clear the air-passages. It may also be useful in the warm bath to direct a stream of cold water against the upper portion of the cervical spine, in order to stimulate the respiratory center to greater activity. After recovery from the disease a **sojourn in the country or in the woods** should be urgently recommended.

FIBRINOUS INFLAMMATION OF THE LUNGS (FIBRINOUS PNEUMONIA).

Etiology.—Fibrinous pneumonia, often designated also croupous pneumonia, is an exceedingly *common infectious disease*, by which almost 4 per cent. of the community are attacked annually. The pneumoniacoccus of Fränkel acts as the carrier of the infection, although it has been maintained that in a small number of instances other bacteria also induce the disease.

The *pneumoniacocci* of Fränkel are somewhat long structures, in shape resembling a candle-flame, which are surrounded by a bright capsule and are arranged usually in pairs, but frequently also in groups of several within a common capsule (Fig. 22). They have, therefore, also been designated *diplococcus pneumoniæ s. lanceolatus*. They occur also in the saliva of healthy individuals, and cause death in rabbits, with septic manifestations, so that they have also been designated *sputum-cocci* or *cocci of sputum-septicæmia*. It is noteworthy that, in contradistinction from the pneumonia-cocci of Friedländer, which resemble them, and which for a time were erroneously considered as the exciting agents of fibrinous pneumonia, they can be stained by Gram's method.

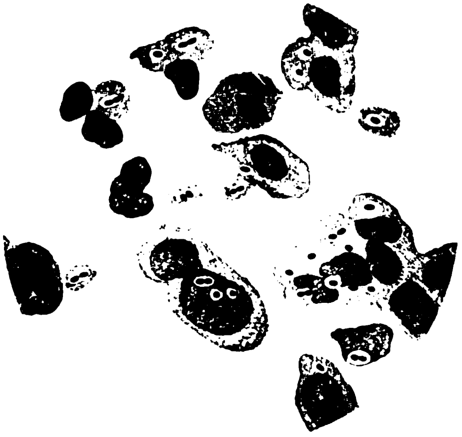

FIG. 22.—Pneumoniacocci of Fränkel from pneumonic sputum. Gentian-violet stain: oil-immersion; magnified 1000 times (personal observation, Zurich clinic).

Although infection with the pneumoniacocci of Fränkel is essential for the development of fibrinous pneumonia, there are certain conditions that favor infection of the lungs—so-called **contributory causes of the infection.** Among these *exposure to cold* occupies the most conspicuous place, while *traumatism* and *preceding infectious disease* are less commonly operative. In overcrowded barracks, prisons, and reformatories, extensive epidemics of pneumonia have at times occurred, obviously because the air was contaminated and filled with pneumoniacocci. *Transmission of the disease from one person to another* or *through the intermediation of a third person* has also been observed. Frequently, however, no contributory cause can be elicited, and the condition is then designated a *genuine* or *primary fibrinous pneumonia*. Like most infectious diseases, fibrinous pneumonia also occurs *sporadically* and *epidemically*. Epidemics occur especially in the spring months, from March to May. Experience has shown that *men are attacked more commonly than women*. Individuals debilitated by disease, advanced age, or alcoholism exhibit a more marked predisposition to the disease than others. The disorder spares no *period of life*, and not rarely attacks children. There is an undeniable tendency to *repeated attacks*, so that some persons pass through more than twenty attacks of fibrinous pneumonia in the course of their lives.

Anatomic Alterations.—The inflammatory alterations of fibrinous pneumonia do not occur in numerous lobular areas like those of catarrhal pneumonia, but, as a rule, involve the entire lobe of a lung, or still more, whence the designation *lobar pneumonia*. It is customary to distinguish several stages of the inflammatory process, and to designate these congestion, hepatization, and resolution.

In the **stage of inflammatory congestion** the pulmonary capillaries are dilated and tortuous. Blood-plasma is thrown out into the pulmonary alveoli; at the same time round cells and also red blood-corpuscles accumulate in the alveoli, while the alveolar epithelial cells become detached and are in part desquamated.

In the **stage of hepatization** the lung presents a characteristic appearance. On removal of the organs it is noteworthy that in the area of inflammation the impression of the ribs upon the surface of the lungs is visible, because the lungs have obviously increased in volume. The pleura probably is unexceptionally clouded, and covered with fibrinous membrane, if, as is the rule, the inflammation has extended to the surface of the lungs. The condition is thus usually one of *fibrinous pleuropneumonia*. On palpation the lung yields the impression of an airless, solid structure. On section its granular surface is striking, suggesting the appearance of a section of liver, whence the name hepatization. The granular condition is due to coagulation of the primarily fluid alveolar exudate. For this reason the hepatized portions of lung present in the aged and in emphysematous patients with large pulmonary alveoli a coarsely granular, and in children with small pulmonary alveoli a finely granular, appearance. The mucous membrane of the supplying bronchial tubes is reddened and swollen, and the fibrinous inflammation has not rarely extended to the smaller bronchial tubes, so that they contain bronchial casts or fibrinous coagula. Less commonly the larger bronchial tubes also are filled with fibrinous casts, and the condition is then designated *massive pneumonia*. At the outset the hepatized pulmonary tissue exhibits a red color—*stage of red hepatization*. The more, however, the over-distention of the pulmonary blood-vessels subsides the more does the tissue present a grayish color—*stage of gray hepatization*, which ultimately gives place to a yellow color—*stage of yellow hepatization*, as soon as active fatty degeneration takes place in the inflammatory products. As a rule, the inflammation develops in different portions of the lungs at different times, so that generally various stages of hepatization are present side by side. In consequence the lung acquires a mottled appearance, which has not inappropriately been designated as marbled or granite-like. This appearance is especially marked when the diseased lung contains a good deal of melanin.

In the **stage of resolution** liquefaction of the coagulated alveolar exudate takes place in conjunction with mucoid and fatty degeneration, perhaps also in consequence of digestion by bacteria, the liquefied masses are expectorated or absorbed, the pulmonary alveoli again receive air, and gradually the lung resumes its normal appearance.

On *microscopic examination of the lungs* during the stage of hepatization the pulmonary alveoli will be found filled with a fibrinous network, in the midst of which lie round cells, red blood-corpuscles, and alveolar epithelial cells. The fibrinous network is especially dense at the periphery of the pulmonary alveoli, while the cells are more numerous toward the center of the alveoli. Pneumoniacocci are present, partly free, partly enclosed in cells. In the stage of resolution the fibrinous network undergoes disintegration into small granules, and active fatty degeneration takes place in the round cells and the alveolar epithelium.

Fibrinous pneumonia occurs most frequently in the lower lobe, particularly on the right side, and least commonly in the right middle lobe. At times inflammatory foci are present in both lungs—*pneumonia fibrinosa duplex.* If under such conditions corresponding lobes are not involved on both sides, for instance, the right lower and the left upper lobe, the condition is designated *pneumonia fibrinosa cruciata.* If all the lobes of one lung are involved, the condition is known as *pneumonia totalis.*

The situation of pneumonia most commonly in the lower lobe of the right lung is explained by the fact that foreign bodies (pneumoniacocci) more readily enter this lung through its larger bronchus, and on account of its greater aspirating force, and that these by reason of their weight readily find their way into the lower lobe.

Persons dead of fibrinous pneumonia usually exhibit signs of **suffocation and of constitutional infection.** The right heart and the veins are generally filled with blood, the heart with ham-fat clot and masses of coagula, while the left heart contains no blood. The muscles of the trunk and the extremities appear dry and of the color of ham, and exhibit, on microscopic examination, granular turbidity, fatty and vitreous degeneration of the muscle-fibers. Similar histologic alterations are present in the fibers of the heart-muscle. The liver-cells, the parenchymatous cells of the gastric and intestinal glands, the epithelial cells of the uriniferous tubules, and the parenchymatous cells of the pancreas exhibit granular cloudiness and fatty degeneration. *Swelling and hyperemia of the bronchial glands* are constant manifestations of fibrinous pneumonia, and result from absorption of the inflammatory agents from the diseased lung. The *spleen* is frequently but not constantly enlarged, and of diminished consistency (acute infection-

Symptoms.—Fibrinous pneumonia belongs to the class of *cyclic acute febrile infectious diseases*, as it pursues a typical course, and terminates usually within two weeks, with manifestations of a crisis. Two groups of phenomena must be distinguished: In the first place, the **local manifestations**, and in the second the **febrile constitutional manifestations.** The latter are probably due to the entrance into the circulation of toxins generated by the pneumoniacocci. The *period of incubation*—that is, the interval between the occurrence of infection and the development of the first symptoms of the disease—appears in many instances to be only a few hours, although it may be from two to four days, and at times perhaps still longer. As a rule, the disease sets in suddenly, with a *rigor*, which may occur while the patient is engaged at work or is sound asleep. This is followed by a continued *fever*, the temperature rising to 39° or 40° C. (102.2° or 104° F.) and above. The pulse is accelerated, full, and often dicrotic. The face is vividly flushed. The tongue is covered with a whitish or grayish coating. While the thirst is greatly increased, the appetite is entirely wanting. The urine is voided in small amounts, is highly concentrated, and not rarely contains small amounts of albumin (febrile albuminuria). After from five to seven days, at times, however, also earlier or later, profuse sweating suddenly appears upon the skin, most frequently during the evening hours. The patient falls into a deep, quiet sleep, and in the course of

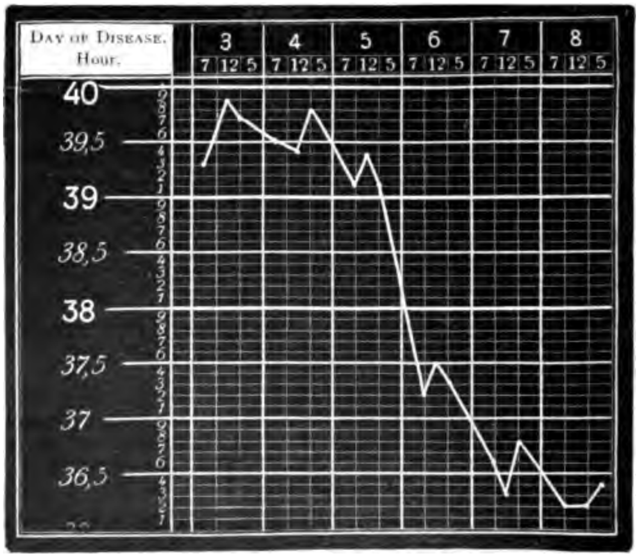

FIG. 23.—Typical temperature-curve of fibrinous pneumonia in a man eighteen years old (personal observation, Zurich clinic).

[illegible]

[illegible]

The [illegible] monia exhibit a [illegible] set in earlier, and [illegible] the first [illegible] the chest, cough, and [illegible] with the stage of the [illegible]

In the *stage of* [illegible] tory area is *tympanitic* [illegible] *impaired*, because the [illegible] and the lung has been [illegible] or *crackling* (fine moist [illegible] alveolar walls separate from [illegible] tion is usually mucous [illegible] streaks of blood.

In the *stage of* [illegible] *percussion-note is* [illegible] *breathing* is heard. A [illegible] absence of air from the [illegible] audible in the inflammatory [illegible] sonating) character. *B*[illegible] *egophony* is present. *R*[illegible] cially significant [illegible] certainty the stage of [illegible] name implies, the sputum ex[illegible] rust, and also a vitreous [illegible] viscid as to be comparable [illegible] vessel the sputum remains [illegible] be cut. On microscopic exa[illegible] and desquamated alveolar epi[illegible] found, which are usually [illegible] the alteration in whose hem[illegible] preparations stained with ani[illegible] Fränkel can be demonstrated. F[illegible] rolled-up grayish or whitish [illegible] which on agitation in water [illegible] delicately branched *fibrinous bronchial* [illegible] and which indicate participation of the smaller bronchial tubes in the fibrinous inflammatory process in the pulmonary alveoli. As fibrinous pneumonia approaches the stage of resolution the sputum acquires a lemon-yellow or saffron-yellow hue, *sputum croceum*, and with the termination of the inflammatory process it acquires the appearance of colorless mucous expectoration.

In the *stage of resolution* the physical alterations in the lungs resemble those present in the stage of congestion. The dulness grows gradually less and less, and the percussion-note becomes at first tympanitic, then clear. The respiratory murmur again resumes its vesicular character, and, above all, *crepitant râles, rhonchus crepitans redux s. crepitatio redux*, are audible in abundance, and which disappear again only when the pulmonary alveoli no longer contain fluid exudate. Patients with pneumonia usually exhibit a reddened and often also a cyanotic hue of the face. At times the redness is more actively developed upon the side of the face corresponding to that of the diseased lung. Generally the patient assumes an *elevated dorsal decubitus.* Recumbency upon the diseased side is, as a rule, avoided in consequence of the pain induced by pressure. *Respiration* is accelerated, partly in consequence of the fever and partly in consequence of diminished participation of the diseased portion of the lung in the respiratory act. The *pulse* is generally accelerated out of proportion to the febrile elevation of temperature. This is believed to be due to the increased blood-pressure in the distribution of the pulmonary artery. The *respiratory movements* appear less active and retarded upon the diseased side, and often jerky in consequence of pleuritic pains. Speech is usually interrupted at short intervals and jerky.

As in most infectious diseases, so also in fibrinous pneumonia a distinction must be made between *anomalies, complications*, and *sequelæ*. The *anomalies of pneumonia* depend at times upon the *severity of the local and constitutional infection*, and at times upon *individual conditions*. The *abortive*, the *ephemeral*, and the *afebrile* forms of pneumonia must be considered as instances of especially mild local and constitutional infection. In cases of *abortive pneumonia* the inflammatory process advances only to the stage of congestion and then undergoes involution. *Ephemeral* or *one-day pneumonia* passes through all the stages of the inflammation, but the fever subsides by crisis within twenty-four hours. *Afebrile pneumonia*, notwithstanding well-developed local manifestations and characteristic expectoration, is a rare condition.

The *protracted*, the *progressive*, and the *erratic* forms of pneumonia must be looked upon as instances of severe local infection. *Protracted pneumonia* persists at times, with fever, for from four to six weeks, and generally terminates with gradual defervescence (lysis). At times it is associated with septic manifestations (multiple articular swellings and the like). *Progressive pneumonia* is characterized by extension of the inflammation from one lobe to an adjacent one, so that finally an entire lobe from an originally small area. *Erratic* or *wandering pneumonia* is attended with the formation of inflammatory foci that develop successively now in one, then in another lobe, but separated from one another by a considerable distance. Under such conditions the disease may persist for many weeks. Not rarely it is attended with multiple articular swellings.

Constitutional infection of especial severity attends *asthenic pneumonia*, which has also been designated typhoid, putrid, or malignant pneumonia. The patients attract attention from the outset on account of the marked asthenia, and, from the presence of delirium, of sordes on the lips and the gums, dryness of the tongue, distention of the abdomen, enlarged spleen, and diarrhea, are suggestive of the clinical picture of typhoid fever. At times slight jaundice develops, whence the name of bilious pneumonia. The sputum frequently is noteworthy on account of its bloody appearance. Death often occurs from asthenia. In many epidemics the asthenic varieties of pneumonia predominate, and are responsible for the high mortality. The asthenic character of fibrinous pneumonia may, in addition to the severity of the general infection, be further dependent upon *individual conditions*. This may find expression particularly in the *fibrinous pneumonia of drunkards and the aged*. Under such circumstances the condition has been spoken of as a *secondary* or *individual asthenic pneumonia*. Drunkards and the aged display but little resistance to infection with pneumoniacocci, are frequently attacked by fibrinous pneumonia, and often lose their lives in consequence. The danger resides especially in paralysis of the heart, which sets in early and often with surprising rapidity.

The *fibrinous pneumonia of children* not rarely exhibits the peculiarity that it sets in with vomiting and eclamptic convulsions instead of with a chill. As in the case of the aged, children generally do not expectorate, but usually swallow the sputum. The fever often terminates gradually, by lysis.

The *recurring* and the *intermittent forms of pneumonia* may be mentioned as special anomalous forms. The former is characterized by the renewed appearance of fever and of inflammation of the lung, the latter involving the area first attacked or others; while the intermittent variety appears in malarious regions under the influence of the malarial parasites (plasmodia), and is characterized by regularly recurrent febrile movement and inflammatory manifestations. It is further distinctive that intermittent pneumonia, which is by no means unattended with danger, can be cured by administration of quinin.

Among the *complications* of fibrinous pneumonia *fibrinous pleurisy* and *bronchial catarrh* are observed so constantly that
an scarcely be considered complications. A pleuritic fric-
nur is heard not at all frequently, and the occurrence of

severe pain in breathing and on coughing must often be sufficient to indicate the existence of pleurisy. By no means rarely pleurisy with effusion occurs, and the exudate is more frequently serous than purulent. The exciting agents of the inflammation are at times pneumoniacocci, at times pyogenic cocci. The same statement is applicable to the remaining inflammatory complications, and pneumoniacocci have been frequently found in different organs, and also in the blood.

One of the most common and most serious complications is indicated by the occurrence of *prune-juice-like expectoration*, which, in contradistinction from rusty-colored sputum, is diffluent, frothy, and dark blackish-red, and in appearance is suggestive of prune-juice. The patients at the same time exhibit marked cyanosis, accelerated and embarrassed breathing, and tracheal râles. On auscultation of the lungs small moist râles are heard over a wide distribution, because the alveoli are filled with serous fluid. Usually the condition is probably one of hypostatic edema, which develops in consequence of enfeeblement of the left ventricle. The more vigorous right ventricle fills the lungs with blood, which, by reason of the weakness of the left heart, does not find sufficiently ready access to the left auricle. The danger of intercurrent pulmonary edema is especially great in persons with an already enfeebled heart (as, for instance, in the aged and in drunkards), and the rapidity with which the condition develops and terminates life is often surprising.

Inflammatory alterations of the endocardium, the myocardium, or the pericardium occur occasionally, but are on the whole of minor significance. The same statement applies to *gastric and intestinal manifestations*. At times *jaundice* appears, and the elimination of biliary coloring-matter in the expectoration gives this a grass-green appearance.

Albuminuria and tube-casts in the urine are common, while hematuria and other signs of acute nephritis are less common. The rapidity with which these phenomena disappear is at times remarkable, even when no noteworthy changes have taken place in the course of the fever and in the local phenomena in the lungs.

Not rarely *delirium* occurs in cases of fibrinous pneumonia, and with especial frequency in children and in drunkards. The greater the elevation of temperature the more likely is delirium to occur, although this may be present also when the fever is of lesser degree, so that it must then be looked upon as a result of the general infection. At times *purulent cerebrospinal meningitis* develops, especially at such times as epidemic cerebrospinal meningitis prevails in association with pneumonia or has prevailed shortly before. Often it is impossible to recognize the disease because characteristic symptoms (rigidity of the neck, inequality of the pupils) are wanting.

Herpes is an unusually common occurrence. It is most frequently situated upon the lips at one side and less commonly on both sides. *Multiple articular swellings* are suggestive of septic states, as is also *purulent parotiditis.*

Among the *sequelæ* of fibrinous pneumonia diseases of the lungs themselves are by far the most common. Among these pulmonary abscess, pulmonary gangrene, pulmonary contraction, and pulmonary tuberculosis may be especially mentioned.

Occasionally *psychopathy*, *neuritis*, and *poliomyelitis* have been observed in the sequence of pneumonia.

Diagnosis.—In the recognition of fibrinous pneumonia the **rusty-colored sputum** is of great diagnostic significance. In cases of central pneumonia it is almost the only certain sign. The **saffron-colored and the prune-juice-like expectoration** also are extremely valuable phenomena in reaching a decision as to the course of the disease. Besides, the typical temperature-curve (continued fever, beginning with a chill, and usually terminating between the fifth and the ninth day by crisis) at times, but usually subsequently, clears up the nature of obscure febrile disease. If doubt arise whether a case be one of fibrinous pneumonia or of typhoid fever, it should be recalled that the presence of **herpes** is opposed to and that of roseola is in favor of the existence of typhoid fever. A positive result with the Widal reaction and the demonstration of typhoid-bacilli in the stools, the urine, or the blood would be decisive for typhoid fever. Peripheral fibrinous pneumonia is readily recognizable from the **manifestations of acute infiltration of the lungs** (dulness, bronchial breathing, increased vocal fremitus, and râles). Although pleurisy with effusion gives rise to dulness on percussion, the vocal fremitus is diminished, and bronchial breathing induced by compression of the lung appears enfeebled. The vocal fremitus is diminished only in cases of massive pneumonia, as in pleurisy, because the occlusion of the bronchial tubes with fibrinous coagula prevents the transmission of the voice-waves from the larynx to the thoracic wall. Nevertheless, in the presence of pleurisy the adjacent organs are frequently displaced; and if effusion takes place into the left pleural cavity, the semilunar space is diminished. It is noteworthy that pleurisy but rarely sets in with a chill, and pursues its course with continued fever. As opposed to pleurisy with effusion would be dulness confined to the upper portion of the thorax, diminishing in intensity from above downward, and exhibiting a most irregular upper boundary. In doubtful cases exploratory puncture will clear up the diagnosis. *Bacteriologic examination of the sputum* for the presence of pneumoniacocci has little bearing upon the diagnosis, because these organisms are also found in the saliva of healthy persons.

It is further important *correctly to localize* diagnosed pneumonic areas. On the right anteriorly the entire area from the apex to the fourth rib cor-

responds to the upper lobe, that between the fourth and the sixth rib to the middle lobe. On the left only the upper lobe is accessible anteriorly. Posteriorly, all alterations down to the level of the spine of the scapula must be referred to the upper lobe, and below this level to the lower lobe on both the right and the left side. Laterally, the fourth rib on either side constitutes the boundary between the upper and the lower lobes. On the right side, however, a process of the middle lobe is situated between the sixth and seventh ribs. Further, variations in the limits named occur to the extent of an intercostal space in consequence of increase in size of a diseased pulmonary lobe.

Prognosis.—The prognosis of fibrinous pneumonia depends upon the **age**, the **constitution**, the **extent**, and the **seat of the inflammation**, the **severity of the general infection**, and the **complications**. Military surgeons report that they rarely see soldiers die in consequence of fibrinous pneumonia, because the patients are robust and youthful individuals. On the other hand, the disease is an unusually serious one in the *aged*, as the majority succumb. Fibrinous pneumonia is also a serious disorder in children. Of especial importance in its bearing upon the course of the disease is the *condition of the heart*. All persons with enfeebled and readily exhausted heart-muscle readily perish in consequence of pulmonary edema or cardiac paralysis. Excessive demands are made upon the functional activity of the heart-muscle by reason of the fever (augmented contraction), and from increased blood-pressure in the pulmonary circulation (from compression of the pulmonary capillaries within the inflammatory area), to which the organ is unable to respond. In the aged also the frequency of death following pneumonia depends principally upon the cardiac enfeeblement. The same statement is applicable to drunkards, to individuals with kyphoscoliosis, obesity, heart-disease, and to pregnant women. Similar conditions may arise in the absence of demonstrable anatomic alterations in the heart-muscle. At times the continuance of life is rendered impossible from the *extent of lung-tissue involved in the inflammatory process*, so that suffocation results. *Fibrinous pneumonia of the upper lobe* is not without justice considered more unfavorable, because it undergoes absorption more slowly and less completely, and is not rarely followed by pulmonary tuberculosis. The significance of the *general infection* is apparent from the seriousness of primarily asthenic pneumonia. To indicate the bearing of *complications* it should suffice to point out that an intercurrent purulent meningitis renders the prognosis unfavorable.

Treatment.—No *specific remedy* for fibrinous pneumonia is yet known. Treatment with **blood-serum** has been attempted of late, but this has scarcely yet led to encouraging practical results. In cases of fibrinous pneumonia in young, robust individuals **expectant treatment** may be pursued as long as serious complications do not arise. When possible, the patient should occupy for himself a quiet and capacious room, whose temperature should be kept at 14° R. (63.5° F.). The air should be kept moist by means of vessels of water, which in winter are placed in the stove-pipe or upon the stove, or water is vaporized by means of a Siegle steam-apparatus, or a solution of carbolic acid (2 per cent.) is sprayed throughout the room three or four times daily. Adequate ventilation may be obtained by opening the windows in an adjoining room. The patient should be given only a liquid diet, especially boiled milk, milk with a little coffee or a little tea, bouillon with egg, and, if thirst be marked, lemonade or white wine, diluted half with water. **Symptomatic treatment** will be resorted to when individual symptoms are distressing or threatening. *Expectorants* are frequently employed when an abundance of moist râles indicate the presence of fluid secretion in the air-passages; for instance:

℞ Infusion of ipecacuanha-root,	0.5 : 180	(7½ grains to 5¾ fluidounces);
Simple sirup,	20.0	(5 fluidrams).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours.

For severe *pain dry* or *wet cups* may be employed. If signs of *cardiac weakness* appear, stimulants and heart-tonics should be resorted to; for instance:

℞ Powdered digitalis-leaves,	0.1	(1½ grains);
Benzoic acid,	0.3	(4½ ");
Camphor,	0.05	(¾ grain);
Sugar,	0.5	(7½ grains).—M.

Make 10 such powders.
Dose: 1 powder every three hours.

Under such conditions strong *wine*, *champagne*, or *cognac* may also be employed. In the presence of *pulmonary edema venesection* may be recommended in addition to the remedies mentioned, while but little success can be hoped for from *bandaging the extremities*. In cases of *fibrinous pneumonia in drunkards alcohol* must be given in sufficient amount in order to avoid the development of delirium tremens and cardiac weakness. Only rarely is the fever persistently so high that resort must be had to *antipyretics*, among which phenacetin, 1.0 (15 grains), is to be preferred.

INTERSTITIAL PNEUMONIA (CIRRHOSIS OF THE LUNG).

Etiology.—Interstitial pneumonia may occur in an *acute* or a *chronic form*. *Acute interstitial pneumonia* is attended with suppuration in the interlobular pulmonary tissue, so that the individual infundibula and alveoli are separated from one another by collections of pus, and the lung in the inflamed areas resembles a corrosion-preparation. The disorder is, therefore, designated *dissecting pneumonia.* As the disease is not recognizable during life, it is without clinical significance. *Chronic interstitial pneumonia* is usually a secondary disorder, generally following diseases of the bronchi, the lungs, or the pleura. It develops with especial frequency in the sequence of *chronic bronchial catarrh,* whether this exist alone or have given rise to bronchial dilatation. *Catarrhal* and *fibrinous pneumonia* also at times give rise to interstitial pneumonia, the former especially when it develops in the sequence of infectious diseases (whooping-cough, measles). *Abscesses, new-growths, echinococci,* and *tuberculous processes in the lungs* also frequently give rise to interstitial pneumonia. Very extensive hyperplasia of the pulmonary interstitial connective tissue is encountered in the diseases due to *inhalation of dust, pneumonokonioses. Pleurisy* is often the cause of the morbid alterations, particularly when the disease has pursued an insidious course, and has given rise to dense and extensive pleural adhesions. It is still a matter for discussion whether *primary chronic interstitial pneumonia* occurs. Among the causes assigned for such a condition are advanced age, alcoholism, and malarial influences.

Anatomic Alterations.—Interstitial pneumonia either leads to diffuse connective-tissue hyperplasia or gives rise to the formation of dense connective-tissue nodules in the lungs. At times both varieties of alteration are associated. The connective tissue is at times of a whitish, at other times of a grayish, or of a blackish or a slaty color, from the abundant presence of melanin. Slate-colored masses of connective tissue occur at the apices of the lungs with especial frequency, where they often enclose tuberculous and caseous areas, and are designated slaty induration or slaty cicatrization. In general, interstitial hyperplasia of the connective tissue takes place frequently for the purpose of circumscribing and enclosing foci of disease in the lungs [illegible] connective tissue has a [illegible] and it thus [illegible] depressed at the [illegible] connective tissue [illegible] and its surface [illegible] a condition has [illegible] cirrhosis of the lung.

[illegible] connective tissue [illegible] of the [illegible] The former [illegible] alveolar [illegible]

Symptoms and Diagnosis.—[illegible]

2 cm. They then manifest themselves especially by dulness, which is elicitable over small areas only upon light percussion, and by retraction of the borders of the lungs and of the thorax. The respiratory murmur over such areas is either enfeebled, or over areas of more considerable size through which a large bronchus passes it is bronchial. In the latter event vocal fremitus and bronchophony are increased. In the presence of *slaty cicatrices at the apex of the lung* it is important to note differences in the level of the apices, a most valuable means of recognizing tuberculous alterations existing in the lung situated at the lower level. *Unilateral contraction of the lung* gives rise to readily recognizable alterations. The thorax exhibits **unilateral retraction**, participates but little in the *respiratory movements*, and frequently is retarded temporarily in movement. At the same time the **intercostal spaces** are narrowed, the **scapula** occupies a lower level, and its inner border is less closely applied to the wall of the chest, and the **vertebral column** exhibits scoliosis, with its convexity directed toward the healthy side of the chest. On pressure upon the thorax a sense of **increased resistance** is conveyed to the hand. The **level of the apex** is lower, and that of the lower margin of the lung is higher than normal. Accordingly, in the presence of contraction of the right lung, the upper border of the lung is abnormally high (normally at the upper border of the seventh rib in the right mammillary line), while upon the left side the semilunar space begins at an unusually high level (normally the left sixth rib). In conformity with the excessive elevation of the diaphragm the **apex of the heart** will occupy a higher intercostal space (normally the left fifth intercostal space) in the presence of contraction of the left lung, and when the margin of the left lung is retracted considerably outward, pulsatile movements of the exposed pulmonary artery become apparent in the left second intercostal space. Frequently not only a cardiac-systolic protrusion is observed in this situation, but also a cardiac-diastolic vibration. The latter is dependent upon increased unfolding of the semilunar valves of the pulmonary artery, which in turn is dependent upon hypertrophy of the right ventricle, in consequence of obliteration of pulmonary capillaries. At the same time the **movements of the heart** may be visible throughout an unusually great extent. At times the heart undergoes great displacement, and, in the event of contraction of the right lung, it is drawn far into the right half of the chest and with contraction of the left lung out to the left axillary line. The *healthy lung* frequently undergoes compensatory emphysema, which is indicated upon the right by depression of the lower margin of the lung, and upon the left side by diminution in the area of cardiac dulness and of the semilunar space. The respiratory disturbances may be thus so completely neutralized that the patients are scarcely conscious of any distress. The *principal dangers* of the disease

reside either in the causative disorder or in paralysis of the right side of the heart and in conditions of stasis.

Prognosis.—Although the prognosis of interstitial pneumonia is unfavorable in so far as the disorder is incurable, it must not be overlooked that some cases represent natural curative processes or tend to restrict the progress of destructive alterations in the lungs.

Treatment.—Internal remedies afford no relief. **Respiratory gymnastics** should be prescribed, the arm upon the diseased side being raised upon a high object (as a door or a chest), and deep inspirations being made several times daily in order to fill the contracted lung more thoroughly with air, or the healthy lung is for a time restricted in its movement by the application of a bandage in order to stimulate the diseased lung to greater activity.

SUPPURATION OF THE LUNG (PULMONARY ABSCESS).

empyema, pyopericardium, abscess of the liver and of the spleen, and burrowing abscess of the vertebral column. Suppuration of the lung is not a common disorder, and is only exceptionally observed in childhood.

Anatomic Alterations.—In accordance with the number of foci present a distinction must be made between **single** and **multiple abscess of the lung.** Multiple abscesses of the lung occur especially as a result of hematogenous influences and of the aspiration of food. Pulmonary abscesses vary greatly in *size.* At times they are barely visible (*miliary abscess of the lung*), while in other instances they involve the largest part of a pulmonary lobe. Accordingly as the pus lies free in the pulmonary tissue, or is surrounded by a membrane resulting from interstitial pneumonia, the condition is designated a *free* or an *encapsulated pulmonary abscess.* The former is distinguished by an irregular villous wall, the latter by a smooth wall. It may further happen that the encapsulation is incomplete. The *amount of pus* varies, and in addition to the size of the purulent focus, it depends especially upon the fact whether copious expectoration has taken place shortly before death.

Symptoms and Diagnosis.—The most important phenomenon for the recognition of an abscess of the lung is the peculiar **expectoration.** This is *purulent,* diffluent, and abundant, so that it may be as much as 500 c.c. in the course of a day. It usually gives off an acid, buttermilk-like odor. Of especial significance is the occurrence of *shreds of pulmonary tissue,* which on microscopic examination exhibit a gray or a blackish color, as well as the alveolar structure of the pulmonary tissue. They may attain the size of a phalanx of the thumb.

At times *hematoidin-crystals* are found in the sputum, and if the disease pursue a chronic course *cholesterin-plates* also may be present.

Frequently, *expectoration in mouthfuls* is noteworthy. The patients expectorate but seldom, although on each occasion such large amounts of pus are expelled as to be frequently discharged from the mouth and the nose, and, if they in part reënter the esophagus and the larynx, to give rise to vomiting and renewed cough. Of importance also is the **attitude of the patient,** who usually lies constantly upon the affected side, in order that the secretion may collect for a long time before it reaches the mouth of the entering bronchus and excites cough; in the presence of centrally situated purulent foci in the lungs the expectoration may be the only means by which it is possible to recognize the affection, while from the attitude of the patient the lung in which the lesion is situated may be suspected. The *local conditions* found in the lung vary accordingly as the abscess-cavity is filled with pus or contains air, between manifestations of airlessness and those of cavity-formation. The *course of suppuration of the lung* is usually acute. Generally there is fever, which often exhibits a hectic character. The patients attract attention on account of pallor, a tendency to sweating, anorexia, in short, on account of septic manifestations. Death may result from increasing asthenia, or from complications, such as gangrene of the lung and pyopneumothorax. At times a focus of suppuration in the lung ruptures externally, and gives rise to extensive burrowing beneath the skin. Complete recovery may, however, take place, leaving only circumscribed retraction of the chest and dulness on percussion.

Prognosis.—Although the prognosis of suppuration of the lung is always serious, recovery not rarely occurs.

Treatment.—In the first place, an effort should be made to sustain the bodily strength by means of a **nutritious diet.** In addition **balsamics** and **disinfectants** should be employed to prevent suppuration and decomposition of the pus; for instance:

R Oil of turpentine,	10.0 (2½ fluidrams).
Dose: 10 drops in milk thrice daily.	

Or,

R Creosote,	0.15 (2½ minims).
Make 100 such gelatin-capsules.	
Dose: 1 capsule every two or three hours.	

Abscesses of the lungs have in a number of instances been subjected to **operative intervention,** which has resulted in recovery in about 60 per cent. of the cases.

GANGRENE OF THE LUNG.

Etiology.—Gangrene of the lung is characterized by the occurrence first of *mortification* and then of *putrid decomposition of the necrotic pulmonary tissue.* The first alteration results from an enormous invasion by pyogenic cocci, especially the various forms of Staphylococcus pyogenes, while the putrefaction is induced by the putrefactive bacteria (Leptothrix pulmonalis, oïdium, Bacterium coli commune, etc.). Pulmonary gangrene is not a common disorder, and experience has shown that it occurs most frequently in men between the sixteenth and the fortieth year of life. It may be of **bronchogenic, alveolar,** or **hematogenous origin.** At times *putrid bronchitis* extends to the pulmonary tissue, and gives rise to gangrene of the lung. *Aspirated foreign bodies* and *particles of food* also not rarely cause pulmonary gangrene. Sometimes this condition has been observed in the sequence of *carcinoma of the lips and of the tongue,* and of *operations upon the oropharyngeal cavity,* when putrid masses of *[illegible] and disintegrated material* have gained entrance into the larynx, and thence **more deeply into the** bronchial tubes and alveoli, and **have excited inflammation and**

putrefaction in the lungs. At times the exciting agents of disease gain entrance only after preceding *rupture into the bronchial tubes*, as, for instance, in the sequence of carcinoma of the esophagus, burrowing abscesses attending tuberculosis of the vertebræ, suppuration of the bronchial glands, new-growths and suppuration in the mediastinum, carcinoma of the stomach, gastric ulcer, etc. Among the *alveolar causes* fibrinous and less commonly catarrhal *pneumonia* should be mentioned first. Drunkards and debilitated persons are especially exposed to the danger that pneumonia may be transformed into gangrene of the lung. *Abscess of the lung*, *pulmonary tuberculosis*, *carcinoma of the lung*, and *echinococcus of the lung*, may also give rise to gangrene of the lung. Occasionally pulmonary gangrene occurs in the sequence of *injuries to the lung*. Among such factors are not alone gunshot wounds, stab-wounds, and punctured wounds, but also blunt injuries, such as contusions of the thorax, and a long time may elapse before the gangrene supervenes upon the injury. *Hematogenous gangrene of the lung* may be due to *pulmonary embolism*. This occurs especially when emboli are derived from putrid foci, as, for instance, in cases of septicemia, caries of the mastoid process, abscess of the liver or of the brain, gangrene and diphtheria of the skin. Certain *contributory causes* may favor the occurrence of gangrene of the lung. Among these may be mentioned alcoholism, asthenia, over-crowding, and deficient ventilation of living-rooms. Pulmonary gangrene has occasionally been observed to occur endemically under such conditions. Gangrene of the lung occurs not rarely in cases of diabetes.

Anatomic Alterations.—It is customary to make a distinction between *circumscribed* and *diffuse gangrene of the lung*, accordingly as the gangrenous area is sharply demarcated from the healthy pulmonary tissue, or gradually passes into healthy tissue without sharp limitation. The diseased pulmonary tissue at first appears as a friable, brownish, and putrid or offensive-smelling mass, which suggests the appearance of an erosion effected with potassium hydroxid. The tissue gradually undergoes softening, and its products usually find exit externally through a bronchus. If expectorated, it is replaced by a gangrenous pulmonary cavity, which at first exhibits a ragged and villous inner surface. This cavity may be gradually encapsulated by connective tissue, which forms in consequence of interstitial pneumonia, and the formation of granulations upon the inner surface may eventually lead to obliteration of the cavity. The *number* and *the size of the gangrenous areas* in the lungs are subject to wide variation. At times a single area may occupy an entire lobe, or even an entire lung. Usually the gangrenous foci are situated at the surface of the lung, and this is especially true of embolic foci. The bronchi generally exhibit signs of inflammation, and their mucous membrane may also suffer

tructure.

Symptoms, Diagnosis, and Prognosis.—In the recognition of gangrene of the lung the character of the **expectoration** is decisive. In the first place, the sputum often diffuses an *unbearably offensive odor*, which is penetrating and has been compared with that of horse-radish. Often the air of the room is contaminated within the shortest period of time by the fetid odor. In some cases the expectoration loses its offensive odor when exposed to the air, but this becomes again unmistakably apparent as soon as the sputum is vigorously agitated in a vessel. The *amount* of expectoration not rarely reaches 500 c.c. and more per day, and its *consistency* is diffluent. In accordance with its principal constituents the sputum is *mucopurulent*, the purulent masses forming grayish-green opaque globules. The tendency of the expectoration to form *layers* is noteworthy. On standing for some time four layers can be distinguished in the sputum. The uppermost is a frothy layer; the next, one of purulent masses; then follows a grayish-green, thin serous layer; and the lowermost is a crumbling layer of sediment. The expectoration may acquire a reddish-yellow or clay-colored appearance, in consequence of the presence of disintegrated hemoglobin.

In contradistinction from putrid bronchitis the presence of shreds of *pulmonary tissue* in the expectoration is decisive in diagnosis, and these may usually be readily recognized from their smoky-gray color. They may be larger than the extremity of the thumb, and consist merely of exfoliated pulmonary tissue. Their number varies widely. On microscopic examination the alveolar structure of the lungs can be detected. The contained elastic fibers have not rarely undergone destruction, probably in consequence of a digestive process, as ferments resembling trypsin have been obtained from the sputum.

Frequently yellowish or even brownish particles, so-called Dittrich or *mycotic bronchial plugs*, are found in the sediment-layer of the sputum, and which, as has been disclosed on post-mortem examination, form in the bronchial tubes that enter the gangrenous area. If these plugs are compressed, they diffuse a peculiarly disagreeable odor. On microscopic examination they are found to consist mainly of bacteria, partly bacilli, and partly cocci. On addition of tincture of iodin the bacteria largely acquire a violet or bluish color, like the leptothrix-filaments in the mouth, whence the name Leptothrix pulmonalis. Further, only the interior of the leptothrix is stained violet or bluish, while the peripheral membrane appears bright yellow. At times thicker structures are found, with eel-like movement, and also spirilla. Infusoria have also been observed, monas lens and cercomonas. The former appears as a roundish globule with an oscillating flagellum, while the latter is of oblong shape with a tail-like process posteriorly, and one or two flagella anteriorly. In addition to bacteria and infusoria, needles of fatty acids, fat-globules, flakes of pigment, and hematoidin-crystals are also found in the bronchial plugs.

At times expectoration is wanting in cases of pulmonary gangrene, and only the **offensive odor of the expired air** is noted. The condition is to be distinguished from simple fetor of the breath originating in the mouth from the fact that the latter is not appreciable at a short distance from the patient, while the putrid odor of the expired air persists. Not rarely patients have themselves first noticed this odor, which may create an aversion to food and drink. I have seen patients die of pulmonary gangrene who presented no other morbid manifestation than the horribly offensive odor of the expired air.

Individuals with pulmonary gangrene unconsciously prefer the **lateral decubitus upon the affected side of the body,** in order that the fluid secretion may collect in the gangrenous cavity for as long a time as possible before reaching the mucous membrane of the afferent bronchus, and thus induce cough. When the gangrenous area is situated centrally the affected side may be recognized from the attitude of the patient. The condition just described explains why the patients expectorate but seldom, though in great amounts —so-called *expectoration in mouthfuls.*

The *local pulmonary alterations* consist either in signs of infiltration (dulness on percussion, bronchial breathing, ringing râles, increased vocal fremitus and bronchophony) or in signs of a cavity (dull-tympanitic and even metallic percussion-note, bronchial or metallic-bronchial breathing, metallic-ringing râles, increased vocal fremitus and bronchophony, variation in the percussion-note between dull and tympanitic accordingly as the gangrenous cavity contains secretion or after previous expectoration air).

Patients with pulmonary gangrene usually attract attention by reason of their ashen-gray **complexion,** and they rapidly lose strength. Generally the **temperature** is elevated, and the fever may be of hectic type. The **appetite** is, as a rule, impaired, and **diarrhea** readily occurs in those who swallow their expectoration, in consequence of decomposition of the ingested food.

The *course of the disease* may be acute, subacute, or chronic. At times death takes place from progressive asthenia within a few days. Nevertheless, the disease may terminate in recovery, and the signs of a cavity may recede in consequence of cicatrization, which is indicated by retraction of the chest and dulness on percussion. Not rarely improvement and aggravation of the disorder frequently alternate with each other.

Complications are common. Among these one of the most serious is *pulmonary hemorrhage.* The danger of this accident resides especially in the fact that the blood (perhaps in consequence of the presence of ferments in the sputum) undergoes coagulation with difficulty, so that death may result from hemorrhage. At times *pleurisy* is superadded to gangrene of the lung. The fluid exudate may be serous, purulent, or putrid, and in the presence of putrid pleural effusions that apparently occur without appreciable cause suspicion should always be aroused that they may have been excited by a concealed focus of gangrene in the lung. Rupture of the pulmonary pleura will be followed by *pneumothorax*, usually by *hydropneumothorax*. Strangely, the exudate under the conditions last named may be serous. At times *rupture of the gangrenous process* may take place externally, and long fistulous passages may form. Rupture into the mediastinum, into the pericardial cavity, into the esophagus, and through the diaphragm, has also been observed. In some cases *metastases* in distant organs take place through dissemination of pyogenic microorganisms; for instance, abscess of the brain or of the liver. I have also observed multiple painful swelling of the joints.

Among the secondary conditions observed is the formation of *drumstick-fingers*, when pulmonary gangrene pursues a chronic course.

Treatment.—With reference to **prophylactic measures**, attention may be directed especially to the importance of carefully feeding by means of the stomach-tube persons suffering from difficulty in deglutition. The treatment of pulmonary gangrene is based upon similar lines to those that govern that of suppuration of the lungs. A nutritious **diet** is prescribed, and the use of alcohol should be permitted for the purpose of sterilizing sputum that may have been swallowed. If possible the patient should be placed in a spacious **room**, which he should occupy alone, because contamination of the air is injurious and the offensive odor of the expectoration and of the expired air would be extremely disagreeable to other occupants. The offensive odor of the expectoration can to a certain extent be neutralized by the introduction of 1.0 or 2.0 (15 to 30 grains) of naphthalin into the sputum-cup, and covering this with a tightly closing lid. The room must be frequently and thoroughly ventilated. On fine days the patient should be exposed to the open air. Among **internal remedies** *balsamics* and *disinfectants* may be recommended to restrict the secretion and decomposition; for instance:

℞ Oil of turpentine, 10.0 (2½ fluidrams).
Dose: 10 drops thrice daily in milk.

Or,

℞ Myrtol 0.15 (2½ minims).
Make 30 such gelatin-capsules.
Dose: 1 or 2 capsules every two or three hours.

Or,

℞ Creosote, 0.15 (2½ minims).
Make 30 such gelatin-capsules.
Dose: 1 capsule every two or three hours.

It is advisable to place in the room three or four times daily **vessels containing hot water** to which 10 drops of oil of turpentine or of creosote have been added, or in winter upon the stove or in the stovepipe, in order that the patient may inhale the vapor. The employment of **inhalation-apparatus** and **inhalation-masks** is less convenient to the patient, and in my experience does not yield better results. **Operative treatment** of pulmonary gangrene has also been attempted (incision and drainage or thermocautery), and recovery has followed in about 50 per cent. of the cases.

NEW-GROWTHS OF THE LUNG.

Etiology.—Among tumors of the lungs only *carcinomata* and *sarcomata* are of clinical significance. Both varieties of new-growth usually develop *secondarily*. The organ primarily involved is at times remote from the lungs, at times in close proximity, so that under the latter conditions the new-growth extends directly into the lung, as, for instance, from carcinoma of the esophagus, the vertebral column, or the mammary gland. *Primary carcinoma* and *sarcoma* of the lungs are exceedingly uncommon. At times traumatism is assigned as the cause. Primary carcinoma of the lung has been observed frequently in miners in the cobalt-mines of Schneeberg, and has been attributed to the inhalation of dust containing arsenic. Both carcinomata and sarcomata of the lungs occur *more commonly in men* than in women, and they develop principally *after the fortieth year of life*. Sarcomata are considerably less common than carcinomata.

Anatomic Alterations.—*Carcinoma of the lung* appears in two forms, either as an **infiltrating** growth, whose limits are not sharply circumscribed with relation to the healthy tissue, or as a **nodular** growth. The nodules vary in size. At times the lung is filled with innumerable small carcinomatous nodules, which suggest the appearance of miliary tubercles, whence also the designation miliary carcinosis of the lung; while in other instances a single carcinomatous nodule occupies almost an entire pulmonary lobe. Not rarely the nodules project from the surface of the lung as hemispherical tumors, whose centers exhibit a slight depression (carcinomatous umbilication). The number of carcinomatous nodules is, like their size, also susceptible of wide variation. All varieties of carcinoma have been observed in the lungs—the hard scirrhus, the juicy and soft medullary carcinoma, the alveolar carcinoma, and the epithelial carcinoma. Primary carcinoma of the lung arises at times from the epithelium of the bronchial mucous membrane or of the bronchial glands, at other times from the alveolar epithelium. In cases of secondary carcinoma of the lung the elements of the disease gain entrance into the lungs partly through the lymphatics and partly through the blood-vessels. The neoplasm extends by preference from the hilus into the lungs, where it follows the ramifications of the bronchial tubes, advancing especially within the lymphatics of the peribronchial connective tissue. Experience has shown that primary carcinoma of the lung involves most commonly the right upper lobe, while pulmonary sarcoma is more common in the left lung.

Symptoms, Diagnosis, and Prognosis.—The most certain symptom of carcinoma and sarcoma of the lung is the appearance of **tumor-particles in the expectoration,** although this occurs but seldom. At times such particles are demonstrable only microscopically, although in one case of sarcoma of the lung I have found in the expectoration pieces of new-growth as large as the end of the thumb. As may be understood, the expectoration of particles of new-growth can only take place if the tumor has previously undergone softening, and the softened masses have ruptured into a bronchus. Not rarely **hemorrhage** takes place into sarcomata and carcinomata of the lungs. There may thus at times be repeated *hemoptysis,* which becomes serious by reason of its profuseness; or if the blood is retained for some time in the air-passages, in consequence of transformation of the hemoglobin, a raspberry-jelly-like or currant-jelly-like, reddish or brownish-black, or even a greenish material, is expectorated, which is, to a certain degree, distinctive of the affection, though it is also observed at times in cases of pulmonary tuberculosis. The **local phenomena referable to the chest** are variable. In the first place, neoplasms of the lung may conceal themselves behind a *pleurisy.* Under such conditions bloody, fat-containing, and colloid exudates especially are suspicious. On microscopic examination of the fluid obtained by puncture, the presence of fatty granule-cells and of cells with numerous nuclei should be looked for. Miliary carcinosis of the lungs at times strongly suggests the clinical picture of miliary tuberculosis of the lungs. The patients attract attention especially on account of *extreme dyspnea* and *cyanosis,* and death results from suffocation. Tumors of considerable size may be recognized from the presence of *percussion-dulness* of irregular outline. If the entire lung is involved in the new-formation, flatness is obtained everywhere upon the affected side of the chest, and generally *increased resistance* also is appreciable. If the bronchi are not occluded by carcinomatous masses, *bronchial breathing* will be audible. Under such conditions vocal fremitus and resonance are increased. The *chest* is frequently enlarged. Adjacent organs (heart, liver) are displaced. By compression or by extension of the new-growth *pressure-manifestations* are not rarely induced in adjacent organs, among which may be mentioned brachial and intercostal neuralgia, constriction of the esophagus, tracheal and bronchial stenosis, pressure upon the veins of the neck with resulting cyanosis and tumefaction of the skin, and paralysis of the vocal bands. At times the *supraclavicular* and the *axillary lymph-glands* are enlarged and degenerated. The patients suffer mostly from *dyspnea*, and alarming attacks of threatening suffocation often occur. The disease terminates almost unexceptionally in the course of a few months in death, which results in consequence of progressive dyspnea or of marasmus, less commonly from uncontrollable hemorrhage. At times pulmonary gangrene has been superadded to carcinoma of the lung. In rare cases rupture externally has also been observed.

Treatment.—Treatment can be directed only to the amelioration of distressing symptoms. Subcutaneous injection of morphin especially will frequently be required. Krönlein in one case removed successfully a sarcoma of the lung by operative means.

ECHINOCOCCUS OF THE LUNG.

Echinococcus of the lung is a rare disease that is acquired by **aspiration of the ova of the Tænia echinococcus of the dog.** The echinococcus-cyst may have developed *primarily* in the lung, or have invaded the lung *secondarily* from other organs, as, for instance, in the presence of echinococcus of the heart, by embolism, or of echinococcus of the liver following rupture through the diaphragm. The echinococcus-cyst is almost always *monolocular*. The condition consists in the development of a vesicle with milk-glass-like walls, containing clear fluid, and frequently also daughter-cysts. At times suppuration occurs in an echinococcus-cyst, and in consequence of inspissation a putty-like mass forms. Most frequently echinococcus-cysts have developed in the right lower lobe. In size they may become as large as a man's head. *Multilocular* echinococcus of the lung has been observed in only a few instances. The individual vesicles were developed either in the distribution of the pulmonary artery, or in that of the pulmonary veins.

Echinococci of the lungs can scarcely be diagnosed with certainty unless **echinococcus-cysts** or **echinococcus-hooklets** are found in the sputum. The membranes may be readily recognized from their laminated structure, and no difficulty is encountered in recognizing echinococcus-hooklets. When echinococcus-membranes are expectorated they may be replaced by gangrene of the lung.

At times echinococcus of the lung is attended with frequent **hemoptysis**, concerning whose origin doubt still exists. When echinococcus-cysts are seated at the surface of the lung their presence is disclosed by **dulness on percussion** of irregular outline and over which the respiratory murmur, vocal fremitus, and bronchophony are wanting. Frequently the patients complain of dyspnea and of stabbing pain in the side. Echinococcus-cysts have a tendency to grow and to *rupture into adjacent cavities*. Rupture into the bronchial tubes has already been mentioned. Rupture may take place also into the pleural cavity, to which pleurisy, pneumothorax, or hydropneumothorax will be superadded. Rupture may even take place through the chest-wall.

The **prognosis** of echinococcus of the lung is serious. Among other conditions sudden rupture or exploratory puncture may be followed by the entrance of echinococcus-fluid into the air-passages, and death from suffocation.

The **treatment** can only be **surgical**, the cyst being removed by means of the knife or the thermocautery.

ANEURYSM OF THE PULMONARY ARTERY.

Aneurysm of the pulmonary artery is rare, but it may occur even in early life. It gives rise to a diffuse (expansile) pulsating tumor in the left second intercostal space, to cardiac-systolic murmurs, and to dilatation and hypertrophy of the right ventricle. This last condition distinguishes it from aneurysm of the aorta. The patients are usually cyanotic and dyspneic, and suffer a good deal from hemoptysis. The principal danger consists in rupture and fatal hemorrhage. The treatment is like that of aneurysm of the aorta (pages 70 and 71).

VI. DISEASES OF THE PLEURA.

INFLAMMATION OF THE PLEURA (PLEURISY).

Etiology.—Inflammation of the pleura arises through the agency of **bacteria**. Whether such inflammation may occur as a result solely of chemic irritants has not yet been demonstrated. *Various bacteria* may give rise to inflammation of the pleura, though it is by no means invariable to detect bacteria in the pleural exudate. In addition to the widespread exciting agents of inflammation and suppuration (Streptococcus and Staphylococcus pyogenes) specific bacteria have been found in the pleural exudate, among which may be mentioned Fränkel's pneumococci, tubercle-bacilli, typhoid-bacilli, and gonococci. The Bacterium coli commune has also been found in the pleural exudate in numerous instances. All of those influences that were formerly considered as causes of inflammation of the pleura are now looked upon as merely **contributory causes** for the infection. These include *exposure to cold, traumatism, preceding infectious disease, preceding disease of the pleura itself* (*tuberculosis, carcinoma*), and *debilitating diseases* (*carcinoma, tuberculosis, suppuration, chronic disease of the kidneys, chronic disease of the heart, syphilitic cachexia, scorbutus*, etc.). At times inflammation of the pleura arises by extension from adjacent disease, as, for instance, pneumonia, pulmonary tuberculosis, gangrene or abscess of the lung, pericarditis, inflammation of the ribs, peritonitis, etc. In some cases no cause for the inflammation of the pleura can be determined (*cryptogenetic pleurisy*). Under such conditions the pleurisy is usually tuberculous, and frequently secondary to preëxisting tuberculosis of the tracheobronchial lymphatic glands. Pleurisy occurs at all periods of life, but most commonly *between the fifteenth and the fortieth year*. *Men* are attacked more commonly than women.

Anatomic Alterations.—In like manner, as in the case of pericarditis, a distinction is made between **dry** and **moist** pleurisy. *Dry pleurisy* is also known as *fibrinous pleurisy*, because it is attended with the formation of fibrinous deposits upon the pleura. At first these are thin and transparent, so that the smooth and glistening surface of the healthy pleura appears as if clouded with vapor. Later, they increase in thickness, and collect in considerable amount, especially in the interlobular fissures of the lung. If recovery takes place, the fibrinous exudate may undergo complete absorption. Often, however, it undergoes organization into connective tissue, and *pleural adhesions* form, uniting the pulmonary and the costal pleura by connective-tissue bands or membranes. At times the adhesions are so extensive as completely to obliterate the pleural cavity.

Moist pleurisy is characterized by the accumulation of a fluid exudate in the pleural cavity. This may be serous, purulent, or hemorrhagic in nature. Accordingly, a distinction is made between *serous pleurisy*, *purulent pleurisy* (pyothorax, empyema), and *hemorrhagic pleurisy*. Should putrid decomposition take place in the pus through the agency of putrefactive bacteria, putrid pleurisy develops. The inflammatory alterations in a case of moist pleurisy usually begin with the symptoms of a fibrinous pleurisy, and these persist also at the time of fluid effusion. This fact is expressed by the designation serofibrinous pleurisy. The *amount of the exudate* may reach ten liters and more, and it is therefore not surprising that the lung should be compressed until it is airless, while adjacent organs (heart, diaphragm, liver, spleen) undergo more or less marked *displacement*.

Symptoms.—*Fibrinous pleurisy* can be recognized with certainty only in the presence of a **pleuritic friction-murmur.** This may be of varying intensity and extent and of variable duration. At times it is appreciable only on most careful auscultation as a faint rubbing, while in other instances it gives rise to a loud, creaking murmur, which has been designated as the *creaking of new leather*, as it has been compared with the sounds generated by the bending of the sole of a leather shoe. It also resembles the crackling of a firmly compressed snow-ball. Murmurs of considerable intensity can be felt as **pleural fremitus.** At times the patient is conscious of rubbing movements in his chest. The murmur may also be propagated for a slight distance from the patient. At times pleuritic friction-murmurs are audible only within circumscribed areas, not exceeding one or two centimeters in diameter. In other instances they can be heard over almost the entire half of the chest. At times pleuritic friction-murmurs persist for only a few hours. On the other hand, however, they may continue for many weeks and months. Not rarely they disappear after repeated deep inspiratory efforts, because under such conditions the layers of the pleura move smoothly upon one another. After a short time, however, they generally reappear.

Fibrinous pleurisy is usually associated with **pleural pain,** and it is important to determine its extent by means of palpation in order to obtain an approximate idea of the inflammatory area. The pain is augmented considerably on cough and on deep breathing, in consequence of increased rubbing of the inflamed layers of the pleura. In some instances fibrinous pleurisy discloses itself only by pleural pain, and the diagnosis of the disease under such conditions is exceedingly difficult. The pleural pain is responsible for certain **respiratory disturbances.** The respiratory movements are often belated upon the affected side of the chest, are only superficial, and follow at intervals. In harmony with this fact the respiratory murmur is faint and frequently also interrupted (jerky). At times there is marked **dyspnea,** because the respiratory movements cannot take place with sufficient freedom. The pleural pain causes the patient, as a rule, to assume a **passive attitude upon the healthy side of the body.**

Patients with fibrinous pleurisy are often harassed by distressing **irritation of the air-passages** and **cough.** They expectorate little or none at all, and suffer from so-called dry cough. Pressure in an intercostal space is usually followed by cough. Apart from pain and disturbed sleep the *general condition* may remain unchanged. At times, however, febrile movement occurs, but this is usually moderate. Among the *sequelæ* of fibrinous pleurisy pleuritic **adhesions** are especially to be named. These may cause obliteration of the complementary pleural spaces, indicated by respiratory immobility of the lower and median borders of the lungs; or they may cause complete obliteration of the pleural cavity, to which dilatation and hypertrophy of the heart may be superadded in consequence of the impaired aspirating power of the lungs upon the blood-stream.

All forms of *moist pleurisy* agree with regard to their physical alterations. From a diagnostic point of view **dulness on percussion** and **diminution in vocal fremitus** are especially distinctive. We shall now consider in detail the individual methods of examination. On *inspection* it will frequently be observed that the patients prefer to lie **upon the diseased side of the body,** obviously in order that respiratory movement of the free, healthy side of the chest directed upward may be as extensive as possible. The diseased side of the chest participates but little, if at all, in the **respiratory movement,** because the pressure of the pleural effusion prevents distention of the thorax and of the lung. The **circumference of the chest** upon the diseased side is increased, and not rarely the **apex-beat of the heart** is displaced toward the healthy side, and the liver is displaced downward. The *type of breathing* is characterized by active participation of the abdominal muscles. *Palpation* yields important diagnostic information, disclosing **enfeeblement or disappearance of vocal fremitus.** The fluid that has insinuated itself between the surface of the lung and the chest-wall is quite capable of preventing conduction of the voice-waves from the bronchial tree to the chest-wall. If the examination for vocal fremitus be made with the border of the hand, it is possible to determine accurately the level at which the exudate begins, from the diminution in the vocal fremitus. The same information can also be gained by direct palpation of the thorax, because the sense of resistance is increased at the limits of the exudation. On *percussion,* dulness is detected within the limits of the exudate, and becomes the greater as the lower portions of the thorax are approached, that is, as the layer of exudate becomes the thicker. In the majority of cases the upper limit of dulness is highest at the side of the vertebral column, gradually declining toward the side and the front of the chest. This manifestation can be explained by the fact that in the dorsal decubitus a horizontal section passes through the vertebral column at a higher level than through the anterior aspect of the chest, and that the surface of a pleural exudate tends to occupy a horizontal position in the thorax. Pleural adhesions naturally may prevent this arrangement. Probably wavy elevations and depressions in the limits of dulness upon the side of the chest frequently depend upon such adhesions (Damoiseau's curves).

Percussion of the upper intercostal spaces upon the anterior aspect of the chest discloses upon the diseased side frequently a deep-tympanitic note, because the pulmonary tissue here is relaxed. If the pleural effusion is large, the percussion-note in this situation is not rarely dull-tympanitic, and this varies as the mouth is opened and closed—Williams' *tracheal note*—in consequence of vibrations of air in the bronchus.

Auscultation not rarely discloses a **pleuritic friction-murmur** at the upper level of a pleural exudate. In other instances this may be heard only when absorption of the exudate has begun, and the pleural layers covered with fibrinous deposit again come in contact. **The respiratory murmur** may be vesicular, bronchial, or **abolished.** It retains its vesicular character as long as the lung is not compressed sufficiently to expel all of the air. In the latter event bronchial breathing sets in. Over exceedingly thick exudates no respiratory murmur at all is audible. On auscultation of the voice either **diminished bronchophony, or egophony,** is appreciable. The latter is observed especially over small exudates or at the upper level, and beside the vertebral column when larger exudates are present. It depends upon the fact that superficial bronchial tubes are readily compressed, so that the voice-waves for the time being pass beyond the points of compression and reach the surface of the chest. The *influence of pleural exudates upon adjacent organs* is noteworthy. In the presence of right-sided pleurisy the heart is at times displaced so far to the left that its apex-beat may be present in the midaxillary line, while in the presence of left-sided pleurisy the heart may be displaced to the right beyond the right mammillary line. At the same time it should be emphasized that the heart is always so displaced to the right that its apex is directed to the left and outward, so that the pulsating part furthermost to the right corresponds to the right border of the heart. In addition to the heart, the **liver** also is frequently displaced by pleural effusions. Right-sided pleural effusions displace the lower margin of the liver at times below the level of the umbilicus. In the presence of left-sided pleurisy the left lobe of the liver is at times deflected downward at the round ligament. Left-sided pleural effusions, as a rule, cause diminution in size or obliteration of the **semilunar space,** lying below the heart and corresponding to the fundus of the stomach, by their weight pushing the diaphragm and the stomach downward. In the same way the spleen, not enlarged, but displaced, may be accessible to the fingers.

The *general condition* at times suffers surprisingly little in spite of an extensive effusion, and some persons continue their work uninterruptedly, although the entire pleural cavity may be filled with exudate. Others complain of **dyspnea, stitches in the side,** and **cough.** In persons with a purulent exudate pallor and anorexia often develop rapidly. Not rarely **distention of the veins of the neck** and **marked cyanosis** appear. Usually there is febrile movement of varying degree, although the disease sometimes pursues an afebrile course. The **elimination of urine** is generally diminished.

In accordance with the duration of the disease a distinction is made between acute, subacute, and chronic pleurisy, as the disease is protracted for as long as four weeks or eight weeks or more. The designation pleuritis acutissima has been applied to cases in which the attack has set in with a chill, followed by high fever, delirium, meteorism, and enlargement of the spleen, and has terminated fatally, often within a few days. After absorption of a pleural exudate has taken place complete recovery may ensue. Often areas of dulness remain, because considerable thickening—

pleuritic cicatrices—have formed. Under such conditions **unilateral retraction of the chest** readily takes place, especially when the lung has been compressed by the exudate for a long time, so that complete distention of the atelectatic areas in the lung is prevented by adhesions. At the same time displacement of the heart and the liver frequently takes place. The heart is pushed unusually deep into the diseased chest, while the liver often occupies an uncommonly high position. Such conditions fail to develop only when the organs named become fixed by adhesions in the positions to which they were displaced at the time of the exudate. Patients who have recovered from an attack of pleurisy suffer for a long time from *stitches* in the diseased side of the chest, and from *shortness of breath,* conditions that probably depend essentially upon pleuritic adhesions.

The *complications* of pleurisy with effusion depend upon the amount and the nature of the exudate. Large accumulations of fluid in the pleural cavity may by marked compression of the lungs and the heart give rise to *danger of suffocation.* At times *fatal syncope* of sudden onset has been observed, occurring especially on assuming the vertical posture. Various conditions may be responsible for such accidents, especially cardiac weakness and cerebral anemia, and embolism of the pulmonary artery, the emboli being derived from cardiac thrombi in the right ventricle. Much less commonly sudden distortion of the inferior vena cava takes place in consequence of a pleural effusion.

In the presence of purulent pleural effusions *rupture of the pus* may take place externally, or into internal organs. Rupture of pus externally is preceded by bulging of the skin, with fluctuation, the protrusion diminishing in size during inspiration, while it increases in size during expiration, and is capable of extinction on careful pressure. The skin gradually becomes reddened, and finally it may rupture, with escape of the pus, sometimes in a strong stream. The rupture of pus externally is designated *empyema pleuræ necessitatis.* If the condition is left to itself, and this would be a grave mistake, the discharge of pus may cease after a time from closure of the fistula. Renewed accumulation of pus in the pleural cavity may then take place, and rupture again occurs. The patient eventually dies from exhaustion or amyloid degeneration. Empyema necessitatis usually develops in the lateral inferior aspects of the chest, although extensive burrowing at times occurs. *Rupture of purulent pleural effusions into internal organs* takes place most frequently through the lungs. This occurrence will be recognized by the sudden expectoration of large amounts of pus (in mouthfuls), while the exudate in the pleural cavity diminishes. At times the air-passages are so greatly filled with pus that there is danger of suffocation, particularly if the accident occurs during the night. Not rarely the pus within the air-passages acquires a putrid odor, although it may have been odorless in the pleural cavity. The expectoration of considerable amounts of pus may be repeated for a varying length of time, and in varying amount, but, as a rule, the pus constantly reaccumulates in the pleural cavity, and also here threatens to cause death by exhaustion or amyloid disease.

In rare cases the discharge of pus does not take place through a pulmonary fistula of considerable size, but the pus is taken up by the pulmonary tissue as by a sponge and is gradually expectorated in the form of purulent sputum.

Rupture of a purulent pleural effusion into the pericardium, the mediastinum, the trachea, the esophagus, the stomach, the intestine, etc., takes place but rarely. Among the complications of pleurisy *pericarditis* may yet be mentioned, and this, experience has shown, occurs most commonly in association with left-sided pleurisy.

Diaphragmatic pleurisy is a special variety of the disease, which, as its name indicates, involves the diaphragmatic pleura. The patients complain of severe pain in the lower portion of the chest, and there is usually marked tenderness upon pressure at the lower border of the chest. The irritation of the air-passages may be considerable. Often there is painful eructation, and the act of swallowing may be attended with severe pain as soon as the ingesta pass the esophageal foramen. At times frequent vomiting takes place. Jaundice has been observed in cases of right-sided diaphragmatic pleurisy, and it has been attributed to the deficiency in the respiratory movement of the diaphragm, which is thus no longer capable of causing expulsion of the bile from the biliary passages in the liver. Dulness over the thorax may be wanting because fluid may accumulate between the base of the lung and the diaphragm without coming in contact with the chest-wall.

The designation *interlobular pleurisy* is applied to cases in which fluid exudate has collected in the interlobular fissures. The exudate is usually purulent, gives rise to an area of girdle-like dulness around the thorax, and frequently ruptures into the lung. *Multilocular pleurisy* is characterized by the presence of a number of encapsulated foci of exudate. Usually the exudate is of similar character in all of the foci, but it may be that in some the fluid is serous, in others purulent. *Pulsating pleurisy* is characterized by pulsation of the diseased side of the chest, transmitted from the heart to the exudate and the chest-wall. It is almost always associated with a left-sided purulent exudate, though I have observed right-sided pulsating serothorax in a boy. Pulsating pleurisy may be confounded with aneurysm of the aorta, but it is unattended with vascular murmurs.

Diagnosis.—The *recognition of a fibrinous pleurisy* is, as a rule, easy if a **pleuritic friction-murmur** can be heard. This might at best be confounded with *snoring* or *sonorous râles*, but these are largely dependent upon efforts at cough, which may cause their disappearance or material alteration in them. The *differentiation between pleuritic and pericarditic murmurs* is discussed on page 57. Should it be necessary to make a diagnosis of fibrinous pleurisy from the **pleuritic pain** alone, care would be demanded to avoid confusion with *muscular rheumatism* and *intercostal neuralgia*. In cases of muscular rheumatism pain occurs especially when the affected muscles are grasped between the fingers, and in cases of intercostal neuralgia the pain is accurately limited to the course of an intercostal nerve. The *diagnosis of a fluid exudate in the pleural cavity* is based principally upon dulness on percussion, diminution in vocal fremitus, and displacement of adjacent organs. *Massive pneumonia* and *new-growths of the lung* also give rise to dulness and diminution in vocal fremitus, but they are unattended with displacement of the heart and of the liver. The differentiation between pleurisy with effusion and *new-growths of the pleura* may be impossible, but the latter are rare. There is but one certain and harmless mode of determining the *nature of an effusion into the pleural cavity*, namely, *exploratory puncture*, which should be made in as low an intercostal space as possible, by means of a sterile syringe specially designed for this purpose. *Encapsulated pleurisy* at times gives rise to error in diagnosis, for if it be situated near the heart, the spleen, or the liver, it might be mistaken for an enlargement of one of the organs named. In the differential diagnosis especial consideration should be given to the fact that in encapsulated pleurisy the outline of the area of dulness is irregular. Difficulties may further arise in the differential diagnosis between *encapsulated empyema* and *subphrenic abscess*. The diagnosis of an accumulation of pus beneath the diaphragm is based upon the previous existence of disease of the abdominal organs, the unusually high level upon the anterior aspect of the chest of the dulness corresponding to the accumulation of pus, the fecal odor of the pus obtained on exploratory puncture, and the existence in the abscess-cavity of positive pressure in inspiration and of negative pressure in expiration as disclosed by a manometer connected with the cannula of a trocar. *Empyema pleuræ necessitatis* finally may give rise to confusion with cutaneous or muscular abscesses, bone-abscesses of the ribs, and burrowing abscesses of the vertebral column, but under all of these conditions respiratory variations in the external extent of the abscess are wanting.

Prognosis.—Most cases of pleurisy terminate in recovery, and the prognosis is therefore favorable. Unfortunately, however, almost two-thirds of the cases are tuberculous in nature, and in this fact resides the danger that after a time infection and tuberculosis will make their appearance in other organs, most frequently in the lungs. At times exudates are, by reason of their great size, [illegible] inflammation. [illegible]

Treatment.—[illegible]

[illegible] 10 to 15 grains.

[illegible] 10 to 15 grains.

[illegible] influence of salicylic acid and [illegible]. Extensive experi[illegible] and specific results are [illegible] from the use of *derivatives* [illegible] mustard-plasters, [illegible] potassium iodid, applica[illegible] of iodin or of oint[illegible] potassium iodid or of iodo[illegible] pilocarpin, hot-[illegible] digitalis, diure[illegible]. **Operative treatment for serous pleurisy** is indicated only when the effusion is so extensive from the outset that death from suffocation is threatened in consequence of displacement of the lungs and the heart, or when no diminution in the effusion can be recognized after the lapse of four weeks, or when even an increase in the effusion has taken place in the fourth week of the disease. For puncture of the pleural cavity I employ an inexpensive and simple

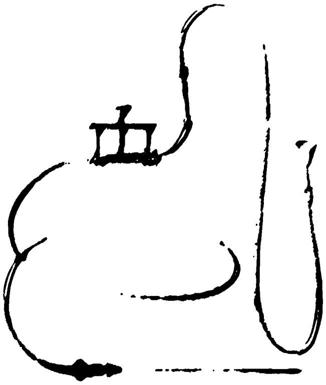

Fig. [illegible] Apparatus for puncture of the pleural cavity.

apparatus consisting of a hollow needle, a rubber tube, which is interrupted at its middle by a glass tube in order that the flow of fluid may be the better observed, and a glass funnel and a clamp (Fig. 24). The apparatus is filled with sterile water and the clamp is closed. After the skin has been thoroughly washed with soap, alcohol, ether, and carbolic-acid solution (5.0 : 100), the needle is introduced into an intercostal space, when the funnel is depressed and immersed beneath the surface of a solution of carbolic acid in a glass vessel one-quarter filled, and then the clamp is released. The pleural exudate will flow into the glass receptacle, while entrance of air into the pleural cavity is impossible. I permit the escape of fluid to continue until the patient begins to cough actively or complains of pain, or the flow ceases spontaneously. Naturally the flow will stop when the pressure in the pleural cavity equals that of the external atmosphere. It is impossible for negative pressure to develop in the pleural cavity.

Rarely in cases of pleurisy negative pressure exists in the pleural cavity before puncture is undertaken. Under such conditions the apparatus described would fail to secure evacuation of the pleural contents, and aspirating instruments would be required, among which may be mentioned the syringe of Dieulafoy, the apparatus of Potain, and the jar of Fürbringer.

Operative intervention is the only appropriate treatment for all cases of *purulent pleurisy,* and I should recommend *resection of ribs and incision.* After puncture is performed fluid usually reaccumulates. *Siphon-drainage* by the method of Bülau has often been employed. With the aid of a trocar a rubber tube is introduced into the pleural cavity, while the other extremity of the tube is immersed beneath the surface of a carbolic-acid solution (5.0 : 100) contained in a suitable vessel. In this way provision is afforded for constant discharge of the pus. Often, however, the formation of pus continues for weeks, and resort must eventually be had to rib-resection and incision. *Putrid pleurisy* requires the same treatment as purulent pleurisy, except that after opening of the thorax and discharge of the pus disinfectant and deodorant irrigation should be practised. In this connection carbolic acid and mercuric chlorid should be avoided, as fatal intoxication may result from rapid absorption of these substances, and zinc chlorid (from 3.0 to 6.0 : 100), aluminum acetate (from 1.0 to 2.0 : 100), or boric acid (2.0 : 100) should preferably be employed. *Hemorrhagic pleurisy* will require puncture only when life is threatened by the large amount of blood present. After recovery from pleurisy has taken place it is often desirable to restore the distensibility of the previously compressed lungs by means of *pulmonary gymnastics.* A succession of deep inspirations while one arm is raised, and repeated several times daily, will suffice. The arm corresponding to the diseased side of the chest should be raised. *Pneumatotherapy* also should be recommended, particularly if the pleurisy is tuberculous in nature. Under such conditions the patients should avoid inhalation of dust and employment in closed rooms.

PNEUMOTHORAX.

Anatomic Alterations.—Any *accumulation of gas in the pleural cavity* is designated pneumothorax. As a rule, fluid also is present in addition to the gas—thus a *hydropneumothorax.* The fluid may be serous, purulent, putrid, or hemorrhagic (*seropneumothorax, pyopneumothorax, hemopneumothorax*). Accordingly as gas and fluid are freely movable in the pleural cavity or are encapsulated by pleuritic adhesions, the condition is spoken of as either a *free* or an *encapsulated pneumothorax* or *hydropneumothorax.* In the cadaver also the existence of pneumothorax and hydropneumothorax is often characterized by unusual **dilatation of the diseased side of the chest** (ectasis), which on percussion yields a deeply resonant note like an empty barrel. On opening the abdominal cavity the **diaphragm** is usually found pushed downward, with its convexity projecting into this cavity, and the liver or the stomach and the spleen in consequence occupying an unduly low position. On *puncture of the thorax* gas generally escapes with a hissing sound, and often diffuses a disagreeable odor of hydrogen sulphid. The **lung** is, as a rule, compressed and airless, being pushed upward and backward at the side of the vertebral column. At times an opening in the pulmonary pleura can be readily demonstrated, through which the air has entered the pleural cavity. In other instances, however, the opening has closed or is concealed beneath fibrinous deposits. Under such circumstances a tube may be introduced into the main bronchus, the lung submerged in water and filled with air through the bronchus, notice being taken of the point at which air-bubbles appear. **Displacement of adjacent organs** (mediastinum, heart, diaphragm, liver, stomach, and spleen) is, further, distinctive of pneumothorax and hydropneumothorax. It results from the same causes as are operative in pleurisy, but generally attains a more marked degree.

Etiology.—The conditions for the development of pneumothorax are provided when the **pleural cavity communicates directly with air-containing organs or with the external air.** This occurs most commonly in connection with *ulcerative destruction in the pulmonary tissues,* especially pulmonary tuberculosis, gangrene, and abscess of the lung, and less commonly echinococcus, tumors, and embolic infarction of the lung. It is noteworthy that at times the lung contains only a few small tuberculous-caseous foci just beneath the pulmonary pleura, which may escape diagnosis, and when they rupture into the pleural cavity the impression may be created that the pneumothorax was of spontaneous development. The same statements apply to *pneumothorax associated with alve-*

olar pulmonary emphysema, in which at times emphysematous vesicles immediately beneath the pulmonary pleura rupture in consequence of insignificant physical exertion or spontaneously during sleep. At times pneumothorax develops in consequence of **injuries of the lungs.** Among these, punctured wounds are less common than excessive expulsive efforts, physical over-exertion, extreme dyspnea, severe cough and the like—conditions attended with such increase in the intrapulmonary air-pressure that rupture of the pulmonary and pleural tissues takes place. Pneumothorax may be caused also by fracture of the ribs, if the extremities of the fractured bone have penetrated the lung. At times pneumothorax is dependent upon **destructive processes in the esophagus,** such as are observed in association with carcinoma, abscess, softening, and unskilful sounding. **Suppuration of the bronchial glands** also may give rise to pneumothorax, if rupture of the pus takes place simultaneously into the air-passages and the pleural cavity. **Destructive processes in the stomach and intestines** (ulceration, abscess, carcinoma) are among the less common causes of pneumothorax. **Injuries of the chest-wall** also (stab-wounds, gunshot-wounds, and the like) are not common causes of pneumothorax, because the margins of the wound usually become closely applied to the cutting instrument. Pneumothorax is at times associated with *disease in the pleural cavity* itself. Thus it may develop in connection with rupture of an empyema of the pleura through the lungs or the chest-wall. Naturally the fistula is generally so constituted that while it permits escape of the pus, it does not permit the entrance of air into the pleural cavity.

It is further maintained that gas may develop spontaneously in *purulent pleural exudates.* The possibility must be conceded, as there are bacilli with gas-generating properties. V. Ziemssen observed transitory pneumothorax develop after *puncture of a pleural effusion,* and he attributed the condition to evaporation of gases from the exudate.

Symptoms.—When pneumothorax develops in the presence of healthy lungs the patient is seized with intense **dyspnea** and a **sense of suffocation.** Breathing is difficult and gasping, the face exhibits an expression of fear, cyanosis develops, and an appearance of suffocation is presented. If, however, the lungs were previously diseased—for instance, in a case of pulmonary tuberculosis—the pneumothorax may develop so insidiously that the condition will be recognized only upon careful investigation. In cases of pulmonary tuberculosis persistent occupation of the same lateral decubitus should arouse suspicion. In addition to dilatation of the chest and displacement of adjacent organs, the metallic quality of the physical phenomena on percussion and auscultation are especially distinctive of pneumothorax, while in the presence of **hydropneumothorax** movable dulness and splashing sounds are superadded. Pursuing the several methods of physical examination in detail, it is first observed upon *inspection* that **dilatation of the chest** (thoraxectasis) is present on the side on which the pneumothorax is situated, because the negative pressure of the lungs has disappeared; and that the diseased side participates but little if at all in the *respiratory movement.* Breathing is visibly embarrassed, and takes place with the aid of the auxiliary muscles of respiration. Often marked **displacement of the heart** toward the opposite side and unusual **depression of the liver** are also appreciable to the eye. The patients lie upon the diseased side of the chest in order that the other lung may be as unrestricted as possible in its function. **Vocal fremitus** is diminished or abolished in the area of the air-accumulation, and, in cases of hydropneumothorax also in that of the fluid accumulation. Only in places where pleural adhesions with the chest-wall exist may it be maintained or even increased. If the pleural cavity contains gas and fluid, **increased resistance** will be readily appreciable at the upper level of the exudate upon immediate palpation of the chest.

Measurement of the circumference of the chest will disclose a difference between the two sides of as much as 10 cm. and more. Tracings of a *transverse section of the chest*, which can be readily obtained with the aid of a piece of pliable lead wire, applied to both sides of the chest exactly at the same level, and then recording the outlines upon paper with a lead-pencil, will disclose distinctly the differences between the two sides.

The **percussion-note** upon the diseased side is unusually deep and loud, the chest-wall being but rarely so greatly distended by the gas that has escaped as to yield a dull percussion-note. As pneumothorax is attended with the presence of gas in a large cavity with smooth walls, metallic percussion-phenomena will be present. As a rule, their development requires special devices —namely, *percussion with plexor and pleximeter*, and *auscultatory percussion.* In the former a metallic plexor or the handle of the plexor is used instead of one of rubber, because percussion by means of hard substances will elicit by consonance the high overtones that give rise to the metallic note. These can be heard with especial distinctness when the stethoscope is applied to the chest while percussion is practised, and in this way auscultatory percussion is employed. Further, the metallic phenomena can be developed distinctly only at certain points. It will be observed on percussion of a free pneumothorax that the boundaries of adjacent organs (heart, liver) are greatly displaced. As long as the opening through which gas has entered the pleural cavity remains unclosed percussion will yield a *cracked-pot sound.* The percussion-note also may vary in pitch accordingly as the mouth is open or closed, becoming higher under the former condition— so-called Wintrich's *change in pitch*—if the opening is in immediate communication with a bronchus of considerable size. If the opening is situated in the chest-wall, the cracked-pot sound will naturally be no longer heard on closure with the finger, and at the same time the percussion-note will become deeper. In cases of *hydropneumothorax* the percussion-phenomena described will be present in the upper portions of the chest corresponding to the accumulation of gas, while in the lower, in which fluid has accumulated, dulness will be present. While in the dorsal decubitus the upper limits of dulness are higher posteriorly than anteriorly, they assume a horizontal level in the sitting posture, and it is especially characteristic of hydropneumothorax that the *limits of dulness vary in accordance with the position of the body.* The cracked-pot sound and Wintrich's change in pitch may also be present in cases of hydropneumothorax as long as the air-fistula remains open, and is situated above the level of the fluid. If by change of position and displacement of the fluid the fistula is temporarily closed, both the cracked-pot sound and Wintrich's change in pitch disappear, and in this way is provided a means of determining the position of an air-fistula.

Biermer has called attention to the fact that in cases of hydropneumothorax the pitch of the percussion-note varies in accordance with the position of the body—so-called Biermer's *change in pitch.* Usually the percussion-note is increased in pitch in the erect posture because the exudate, by reason of its weight, depresses the diaphragm, and the diameter of the air-cavity is thus increased.

The **auscultatory phenomena elicited** in a case of *pure pneumothorax* vary. At times no respiratory murmur at all is heard, and it has, not without reason, been maintained that unilateral dilatation of the thorax, with a deep, loud percussion-note and an absence of respiratory murmur, is indicative of pneumothorax. In other instances metallic bronchial breathing is audible. This is especially distinct, and sometimes is present exclusively during expiration. Bronchial breathing occurs when the lung is compressed to the degree of airlessness, and within the pleural cavity filled with gas it acquires metallic properties through consonance. Should râles develop within the air-passages, they also acquire a metallic character. Even bronchophony, which is diminished in cases of pneumothorax, also exhibits a metallic character.

Hydropneumothorax is distinguished by the occurrence of a *pleural splashing sound* (succussion-sound, Hippocratic succussion), which is generated on movement of the body by the impact of the pleural fluid against the chest-wall. Vigorous agitation of the patient, change in position from one side of the body to the other, rapid assumption of the vertical posture, jumping upon the feet, and the like, are capable of generating the sound in question.

At times the *sound of the falling drop, gutta cadens—metallic tinkling—* may be heard. This results at times on change of posture, as, for instance, in sitting erect, from the dropping of fluid that has accumulated upon the roughened surface of the pleura.

As long, in a case of hydropneumothorax, as the air-fistula in the pulmonary pleura remains patulous, **expectoration** of purulent material **in mouthfuls** may take place. The patients do not expectorate often in the course of the day, but then the sputum is ejected in large amounts, and is not rarely expelled from both mouth and nose, and in part may enter the larynx and excite vomiting. Expectoration takes place only when sufficient exudate has accumulated in the pleural cavity to reach the level of the air-fistula, and discharge into the bronchi has taken place.

The same effect may be induced also by certain positions of the body. As a rule, the patients persistently occupy the same lateral decubitus, in order to breathe with the least possible restriction with the free lung placed above. At times, however, the patient assumes quite extraordinary attitudes in order to prevent for as long as possible the entrance of pleural fluid into the air-fistula and the air-passages.

The *duration of pneumothorax and hydropneumothorax* is most variable. At times death from suffocation results within a few hours after the development of the disease, while in other instances the affection may persist for years. Under the latter conditions the patients may at times be out of bed and indulge in various physical activities. In a case of pneumothorax gradual absorption of the gas, and thus recovery, may take place. Hydropneumothorax is not rarely converted into a simple pleurisy, the gas disappearing and the pleural effusion remaining. In the presence of a purulent or a putrid hydropneumothorax, not treated by operative measures, death may take place from progressive asthenia and septicemia.

Diagnosis.—The recognition of pneumothorax and hydropneumothorax is usually easy. Often **dyspnea and threatened suffocation of sudden onset** will attract attention, and in addition there will be found **dilatation of the chest, absence of vocal fremitus, metallic phenomena on percussion and auscultation**, and in cases of hydropneumothorax also **pleural splashing sound.** To decide the *nature of the pleural effusion* in a case of hydropneumothorax exploratory puncture will be necessary. The **persistence of an air-fistula** in the pulmonary pleura may be recognized from the presence of the cracked-pot sound, and of Wintrich's change in pitch, and the occurrence of expectoration in mouthfuls. At times a valvular fistula may be temporarily closed by the excessive pressure of the gas in the pleural cavity. If, in addition to the accumulation of gas, fluid also is present in the pleural cavity, and if the fistula is situated below the level of the fluid, gurgling murmurs will be audible if gas be removed from the pleural cavity by means of an aspirator. The sounds generated have been designated *pulmonary-fistula*, or *bubbling murmurs.* These result from the entrance of air through the again patulous fistula into the pleural cavity and its passage upward through the effusion. Naturally, the same manifestation may be observed in the presence of an open hydropneumothorax when the air-fistula is situated beneath the level of the pleural effusion.

In accordance with the *character of the air-fistula* four varieties of pneumothorax may be distinguished, namely the open, the closed, the valvular, and the transitional. *Open pneumothorax* is provided with a single opening through which air enters the pleural cavity without obstruction, and can again make its exit. The pressure of the gas does not exceed that of the atmosphere, and the displacement of adjacent organs is, therefore, usually not excessive. The means for recognizing an open air-fistula have already been described. *Closed pneumothorax* is attended with a closed air-fistula. In accordance with the amount of gas present the pressure in the pleural cavity may equal that of the atmosphere, or be less or greater than this. If air be permitted to escape from the pleural cavity, the intrapleural pressure is diminished and remains permanently unchanged, as additional gas cannot enter. The conditions are different in the presence of *valvular pneumothorax*. Here the pleural cavity continues to be filled with gas until the latter by excess of pressure causes closure of the valvular fistula. The displacement of adjacent organs is usually quite considerable. If gas be evacuated from the pleural cavity by puncture, the fistula reopens, and the conditions become the same as before the puncture. An example of the *transitional variety of pneumothorax* is furnished by valvular pneumothorax, which in the presence of a great excess in the pressure of the gas in the pleural cavity acquires the character of a closed, and with a diminution in the pressure presents those of an open, pneumothorax.

Free hydropneumothorax may be confounded with subphrenic hydropneumothorax, and with the stomach distended with gas and fluid. *Subphrenic hydropneumothorax* consists of a cavity filled with pus and gas situated beneath the diaphragm and between this and the liver or the spleen. It is noteworthy that the development of this disorder has been preceded by disease of the abdominal organs (stomach, intestines), that the purulent masses obtained on puncture possess a fecal odor, and that, if the respiratory variations in the pressure of the gases be studied with the aid of a manometer, it will be found that the pressure is increased during inspiration, to be diminished during expiration, whereas in cases of hydropneumothorax the reverse conditions prevail. If the *stomach be greatly distended with gas, and fluid be simultaneously present,* the resonance of the stomach may give rise to metallic phenomena in the lower portion of the left half of the thorax on percussion and auscultation, and on agitation of the body splashing sounds may appear; but these phenomena are at once modified when a tube is introduced into the stomach and the gases are evacuated.

Encapsulated hydropneumothorax gives rise in part to the same manifestations as pulmonary cavities, but over the latter the intercostal spaces are usually retracted, while the vocal fremitus is increased, and succussion-sounds are but rarely appreciable.

Prognosis.—The prognosis in cases of pneumothorax and hydropneumothorax depends upon the **amount of gas** that has escaped into the pleural cavity and the **cause of the disorder.** Quite considerable accumulations of gas in the pleural cavity at times cause death by suffocation in the shortest possible time. Under certain conditions pneumothorax and hydropneumothorax must even be considered as fortunate occurrences, for it is maintained, not without reason, that such events in cases of pulmonary tuberculosis check the progress of the disease.

Treatment.—In cases of *simple pneumothorax* an **expectant attitude** should be assumed for a time. If serious dyspnea be present, alleviation may be sought by means of **subcutaneous injection of morphin**:

℞ Morphin hydrochlorate,	0.3 (4½ grains);
Glycerin,	
Distilled water,	each, 5.0 (75 minims).—M.

Dose: 0.5 (8 minims) subcutaneously.

In cases in which the action of morphin fails, the air in the pleural cavity should be evacuated by **puncture,** a hollow needle and a rubber tube being employed for the purpose. One end of the rubber tube is connected with the needle, while the other is submerged in a vessel containing a solution of carbolic acid (5.0 : 100). In cases of open pneumothorax puncture would be useless, as the air at once enters the pleural cavity through the fistula. In cases of valvular pneumothorax also there is a possibility of renewed accumulation of gas, so that permanent success can be expected only in cases of closed pneumothorax. In cases of *hydropneumothorax,* **rib-resection** and **incision into the pleural cavity** should be practised when the pleural effusion is purulent or putrid. Tuberculosis of the lungs is not a contraindication of operation.

In cases of putrid hydropneumothorax great care must be observed in *irrigating the pleural cavity* as long as the air-fistula in the lung remains open, as the irrigating fluid may fill the air-passages and cause death by suffocation.

Seropneumothorax requires **puncture of the pleural cavity** when danger of suffocation is imminent; but otherwise an expectant attitude should be assumed. Internal remedies exert no influence

DROPSY OF THE PLEURA (HYDROTHORAX).

Etiology.—Hydrothorax is characterized by the accumulation of a serous *transudate* in the pleural cavity, and usually is bilateral. Among the causes are **increased blood-pressure in the venous system** (*hypostatic edema*) and **anemic** and **cachectic states** (*cachectic edema*). *Hypostatic edema of the pleural cavity* depends, as a rule, upon weakness of the right ventricle, such as occurs especially in conjunction with valvular disease of the heart, myocardial disease, disease of the pericardium, of the lungs, and of the pleura. *Local hypostatic edema* occurs but rarely in the pleural cavity when the azygos vein or hemiazygos vein is obstructed by pressure or by thrombosis. *Cachectic edema* develops in cases of nephritis, carcinoma, suppuration of all kinds, profound anemia, and allied conditions.

Anatomic Alterations.—Hydrothorax occurs generally in association with edema of the skin and accumulation of transudate in other serous cavities. The amount of fluid in the pleural cavity may reach several liters, and under such conditions it is considerably greater in one pleural cavity than in the other in cases in which the patient persistently occupies the same lateral decubitus. The fluid is amber-yellow or greenish-yellow in color, and transparent, and contains at most a few flocculi, which consist of desquamated and swollen endothelial cells or of soft fibrin.

At times the fluid presents a bloody appearance and in some cases of jaundice an icteric hue.

The lungs are rendered airless in greater or lesser degree in consequence of pressure.

Symptoms and Diagnosis.—Hydrothorax may develop quite insidiously. Increasing compression of the lungs gives rise to progressive **dyspnea.** When the latter becomes excessive, *suffocation* results. The diagnosis depends upon the demonstration of an accumulation of fluid in the pleural cavity (dulness, enfeeblement of vocal fremitus and of the respiratory murmur). Inflammatory manifestations, such as stitch-like pain in the side and fever, are usually wanting. To distinguish a pleural exudate and a pleural transudate from each other the specific gravity of the fluid should be noted. A figure below 1015 is indicative of a transudate and a figure above 1018 is indicative of an exudate.

Prognosis.—The prognosis depends upon the **nature of the primary disorder.** If, for instance, it is possible to increase the vigor of the heart, the hydrothorax likewise may be made completely to disappear.

Treatment.—The treatment should in the first place be **directed to the correction of the primary disorder.** If the hydrothorax has attained dangerous proportions, it should be relieved **by puncture,** which may be repeated if necessary.

HEMOTHORAX.

Collections of blood in the pleural cavity may be of traumatic origin or result from rupture of aneurysms of the aorta or the pulmonary artery, from ulceration of the aorta, the pulmonary artery, or the pulmonary vein, in consequence of tuberculous or gangrenous destructive processes in the lungs. The blood in the pleural cavity retains its fluidity for a long time, and occasionally it induces secondary pleurisy.

The **diagnosis** depends upon the signs of an effusion of fluid into the pleural cavity, together with symptoms of internal hemorrhage. Besides, respiratory disturbances become manifest when the lungs are greatly compressed.

The **prognosis** depends upon the amount of the hemorrhage, and its susceptibility of control.

Treatment should be directed to the control of the hemorrhage by means of **hemostatics** and to the prevention of asthenia by the use of **stimulants.** Under certain conditions it will be necessary to remove the blood from the pleural cavity as quickly as possible by means of **puncture.**

CHYLOTHORAX.

The accumulation of chyle in the pleural cavity is an exceedingly rare occurrence. The fluid that collects presents a milky appearance, yields an alkaline reaction, contains sugar and a sugar-generating ferment, and microscopic examination discloses the presence of isolated lymph-corpuscles. Care must be taken to avoid confusion with pleural exudates containing fat, such as occur especially in association with carcinoma of the lung. Exudates containing fat present an abundance of fatty granule-cells.

The **diagnosis** of chylothorax can be made only by puncture and chemic examination of fluid found in the pleural cavity. The fluid is derived from the ruptured thoracic duct.

CARCINOMA OF THE PLEURA.

Carcinoma of the pleura is usually secondary, and develops at times in consequence of direct extension from adjacent structures (lungs, mammary gland, mediastinum) and at other times by metastasis from remote organs. While in some cases innumerable miliary carcinomatous nodules are found upon the pleura, in others tumor-masses larger than a fist are present. Often a fluid effusion is present, at times serous, at other times purulent or hemorrhagic, and at still other times colloid or fatty.

The **diagnosis** of carcinoma of the pleura is difficult. Miliary carcinosis is not rarely attended with intense dyspnea and uncontrollable cough. At times carcinoma of the pleura is concealed behind the signs of a pleurisy with effusion. Under such conditions the presence in the fluid obtained on exploratory puncture of many fatty granular cells, and especially numerous multinucleated cells, is significant. Tumor-masses of considerable size cause dilatation of the heart, displacement of adjacent organs, and diminution of vocal fremitus and of the respiratory murmur, just as does pleurisy with effusion, but the former is distinguished from the latter by the irregular outline of the area of percussion-dulness. At times pulsation is imparted to new-growths from subjacent arteries, so that the presence of an aneurysm is suggested, but the diffuse expansile pulsation of an aneurysm is wanting. Numerous complications may be superadded in consequence of pressure upon and constriction of the esophagus and adjacent vessels.

The **prognosis** is unfavorable.

The **treatment** consists in the relief of the most prominent symptoms.

ECHINOCOCCUS OF THE PLEURA.

Echinococcus-cysts occur but rarely in the pleural cavity. They may rupture into the pleural cavity from the lungs or the liver, or, less commonly, they may develop primarily in the pleural cavity. At times they give rise to pleurisy especially purulent in character. They occasion dulness on percussion, with enfeebled vocal fremitus and respiratory murmur, the dulness being characterized by its irregular limits. At times they cause protrusion externally, and finally they rupture in the same direction. If puncture be deemed necessary, it would be important to examine the fluid obtained—which will be found free from albumin—for the presence of echinococcus-scolices, hooklets, and laminated membrane. The patients often complain of stitch-like pain in the side and dyspnea. The condition is only susceptible of treatment by operative measures.

VII. DISEASES OF THE MEDIASTINUM.

MEDIASTINAL TUMORS.

Anatomic Alterations.—Mediastinal tumors include new-growths of all kinds in the *mediastinum*. These are most commonly situated in the *anterior mediastinum*. They are associated with especial frequency with *diseases of the lymphatic glands* (lymphoma, sarcoma, carcinoma). Much less commonly they arise from the *mediastinal connective tissue* (fibroma, lipoma). At times they are dependent upon *disease of the thymus gland* (sarcoma). Sometimes osteomata arise from the *sternum*. *Substernal struma* also is included among mediastinal tumors. In rare instances dermoid cysts and echinococci have been observed in the mediastinal space. At times new-growths extend from adjacent structures —as, for instance, the pericardium—to the mediastinum, where they attain considerable and conspicuous proportions during life.

Etiology.—In many instances no cause whatever for the development of mediastinal tumors can be elicited. In other instances **traumatism** is assigned as the cause. At times enormous enlargement of lymphatic glands takes place in the sequence of *scrofulosis*, *leukemia*, *pseudoleukemia*, and *syphilis*. Experience has shown that mediastinal tumors occur *more commonly in men* than in women. They appear mostly between the twentieth and the thirtieth year of life.

Symptoms and Diagnosis.—Mediastinal tumors are attended principally with two groups of symptoms—in the first place with **abnormal areas of dulness**, and in addition with **pressure upon and displacement of adjacent organs.** Both groups of symptoms may be present together, or one alone may be encountered. *Abnormal areas of dulness* occur most commonly over the manubrium sterni, and it appears noteworthy in this connection that on percussion, in contradistinction from the normal conditions, the manubrium sterni yields a duller note than the body of the sternum. In the presence of mediastinal tumors of considerable size the area of dulness extends far beyond the borders of the sternum, and if the new-growth has invaded the lungs throughout a large extent, the greater portion of the anterior aspect of the chest may yield dulness on percussion. Further, the marked resistance on percussion is often conspicuous. Tumors in the posterior mediastinum give rise to abnormal areas of dulness at the side of the vertebral column. In this situation also the dulness may attain a considerable extent. *Pressure-phenomena and displacement* involve especially the circulatory and respiratory organs and the nerves. At times, however, the chest itself may be distorted in consequence of pressure, and particularly the manubrium sterni may undergo marked protrusion forward. At times the bony portions of the thorax may be eroded, and the new-growth may be situated directly beneath the skin. At the same time it may be the seat of pulsation transmitted from subjacent arteries, but the movement always consists only in simple elevation and depression, and never, in contradistinction from an aneurysm, in expansile pulsation. The heart is often displaced downward and outward by mediastinal tumors, so that the apex-beat may be situated below the left fifth intercostal space, and outside the left mammillary line. Not rarely the increased extent of the apex-beat is noteworthy, resulting from the greater application of the heart to the anterior wall of the chest. Often *marked fulness and* *tortuosity of some cutaneous veins* are noticeable if pressure has been exerted upon the veins. At times such vessels are apparent beneath the skin of the chest, at other times upon one side of the neck and face, at still other times upon one arm. Usually **cyanosis** is associated, and frequently also **circumscribed edema of the skin.** Pressure upon arteries may give rise to *retardation and reduction in the size of the pulse* upon one side of the body. *Cardiac-systolic murmurs due to pressure* may also be present. At times signs of *unilateral or bilateral bronchostenosis* appear. Cyanosis and intense dyspnea are occasionally due to pressure upon the lungs or to extension of the new-growth to the pulmonary tissue. Not rarely *attacks of increased dyspnea* occur, without the development of demonstrable alteration in the objective conditions. Large mediastinal tumors may cause *displacement of the liver and the spleen. Obstruction of the esophagus* also may result from pressure. Frequently *paralysis of the recurrent laryngeal nerve* is caused by pressure. This is at times unilateral, at other times bilateral, or it may be at first unilateral and subsequently become bilateral. Pressure upon the cervical sympathetic gives rise to *pupillary changes.* Unilateral irritation of the sympathetic causes dilatation, paralysis, and contraction of the pupil. Symptoms of *paralysis of the vagus* also occur, and are manifested by unusual slowness of action of the heart (irritation of the vagus) or, in the presence of paralysis of the vagus, by acceleration of the action of the heart. Attacks of spasm of the esophagus, eructation, and vomiting have also been attributed to irritation of the vagus. Irritation of peripheral nerves may give rise to severe *neuralgia.* The *course of mediastinal tumors* is generally chronic and fatal. Death results most commonly from suffocation, though at times from progressive asthenia.

Prognosis.—Mediastinal tumors require a serious prognosis in all instances, although in rare cases the possibility of recovery cannot be excluded, as, for instance, in those of syphilis.

Treatment.—**Causal treatment** is probably applicable only in cases of syphilis, and then consists in the external and internal employment of preparations of iodin and of mercury. Opportunity for *operative intervention* will be but rarely afforded. Accordingly, **symptomatic treatment** alone will usually be available. It often becomes necessary to relieve the sufferings of the patient as much as possible by means of narcotics, especially subcutaneous injections of morphin.

INFLAMMATION OF THE MEDIASTINUM (MEDIASTINITIS).

Inflammation of the mediastinum is one of the rarest of diseases. It occurs either as a *chronic mediastinitis,* with cicatricial thickening of the mediastinal connective tissue, or as an *acute medi-*

astinitis, which may give rise to a circumscribed *mediastinal abscess.* Among causative factors *traumatism, exposure to cold, infectious diseases,* and *inflammation of adjacent organs* are mentioned.

Acute mediastinitis is attended with *septic manifestations* (chills, fever). The patients frequently complain of a sense of pressure and pain beneath the sternum, and not rarely also of difficulty in swallowing. The skin over the sternum exhibits redness and inflammatory collateral edema. Should a mediastinal abscess develop, dulness over the manubrium sterni appears if the accumulation of pus is situated in the anterior mediastinal space. Rupture of the pus may take place internally or externally, and in the latter event the pus may find its way beneath the skin of the neck or to some remote point in consequence of burrowing. At times large blood-vessels are eroded by the suppurative process, and fatal hemorrhage results.

The **diagnosis** often remains doubtful during life. In the first place, antiphlogistics (ice-bag, leeches, inunctions of mercurial ointment) will be employed, and if the pus be not too deeply situated incision may be practised.

The dangers of *chronic mediastinitis* consist principally in involvement, distortion, and angulation of large vessels or the esophagus or bronchial tubes. At times they give rise to paralysis of the recurrent laryngeal nerve.

Treatment is futile.

INTERSTITIAL MEDIASTINAL EMPHYSEMA.

Accumulation of gas in the connective tissue of the mediastinal space may result from rupture of air-containing organs (lung, esophagus, stomach, intestine) into the mediastinum. It occurs frequently in association with interstitial emphysema of the lungs, and is then dependent upon the same causes. The principal manifestations include a clear percussion-note over the sternum, displacement of the heart outward toward the left, and of the liver downward, and crepitating and clacking râles with the movements of the heart. There may be dyspnea and a sense of oppression.

The **treatment** is purely symptomatic.

PART III.

DISEASES OF THE DIGESTIVE ORGANS.

I. DISEASES OF THE MOUTH.

CATARRHAL STOMATITIS.

Etiology.—Like most catarrhal conditions of mucous membranes, catarrh of the mucous membrane of the mouth may be due to **mechanical, thermic, toxic,** or **infectious** causes. It may, in addition, result from the **extension of adjacent inflammation.** Among *mechanical etiologic factors* the sharp edges of teeth may be especially mentioned. *Thermic injurious agencies* include *burns due to hot articles of food. Toxic stomatitis* occurs not only in the sequence of ingestion of corrosive acids, alkalies, and other poisons, but also in drinkers and smokers from the irritation caused by alcohol and tobacco. Catarrhal stomatitis occurs in infants if the mouth is not cleansed or is but imperfectly cleansed of remnants of milk, the fermentation of which then gives rise to irritation of the mucous membrane; or if the cleansing of the mouth is effected by means of coarse cloths and with too much vigor. Under the conditions last named mechanical irritation of the mucous membrane of the mouth also takes place. At times catarrhal stomatitis arises after the internal or external employment of certain medicaments, as, for instance, potassium iodid or bromid, arsenic, and mercury. *Infectious catarrhal stomatitis* is frequently of secondary origin, and develops in the course of other infectious diseases (typhoid fever, measles, scarlet fever, small-pox, syphilis). Little is known with regard to primary infectious catarrhal stomatitis. It may arise, for instance, by inoculation of gonococci upon the mucous membrane of the mouth. *Stomatitis arising by extension from adjacent disease* occurs with especial frequency in the sequence of diseases of the teeth. In infants it accompanies the irruption of the teeth. Catarrhal conditions of the nares, the pharynx, and especially the stomach, frequently give rise to secondary catarrh of the mucous membrane of the mouth.

Symptoms, Anatomic Alterations, Diagnosis, and Prognosis.—In accordance with the course of the disease a distinction should be made between *acute* and *chronic catarrhal stomatitis.* The latter occurs especially in drinkers and smokers. At times the condition is *circumscribed,* at other times *diffuse.*

Acute catarrhal stomatitis is characterized by **redness, swelling,** and **increased heat** of the diseased parts. At times the lymph-follicles of the mucous membrane are greatly swollen, and superficial loss of tissue may take place. The patients complain principally of a **sense of burning** and even of **pain in the mouth,** and infants frequently dig their fingers into the mouth. The latter also not rarely cease nursing in the midst of the act, or they refuse nourishment altogether on account of the pain. The **secretion of saliva** is increased in consequence of reflex irritation. In children the saliva usually pours out of the mouth, often irritating the external integument and giving rise to linear redness corresponding to the course followed by the secretion. In adults, on the other hand, there is increased frequency in the act of swallowing. The **tongue,** as a rule, presents a grayish or yellowish, even a brownish coating, consisting of remnants of food, desquamated and fattily degenerated epithelial cells, bacteria, especially leptothrix-threads, but containing at times also cholesterin-plates and lime-crystals. At the margins of the tongue and also upon the mucous membrane of the cheeks the impressions of the teeth are visible in the form of grooved depressions. Often a disagreeable, **fetid odor emanates from the mouth.** Frequently complaint is made also of a **disagreeable and pasty taste.** At times there is increased **thirst. Elevation of temperature** occurs now and then in infants. The disease often terminates within a few days, and is unattended with serious sequelæ.

In cases of *chronic catarrhal stomatitis* also the mucous membrane presents an altered, and usually a brownish-red color. Often the epithelium is thickened and turbid, so that grayish-white spots form. At times painful fissures occur in the mucous membrane, but apart from these a sense of burning and of soreness in the mouth is often complained of. Alcoholics and smokers not rarely suffer from chronic catarrhal stomatitis throughout life, but the disorder is quite a harmless one.

Treatment.—The first indication is the removal of the causative factors, **causal treatment,** and in addition the mouth should be rinsed after each meal with **potassium chlorate** (5.0 : 200). In infants the mouth should be carefully cleansed after each meal with the solution named by means of a bit of linen. Applications of **mercuric chlorid** (0.5 : 50) or argentic nitrate (1.0 : 50) **have**

ULCERATIVE STOMATITIS.

Symptoms, Anatomic Alterations, Diagnosis, and Prognosis.—Ulcerative stomatitis is characterized by gangrenous destruction of the mucous membrane of the mouth, especially that of the gums. The disease begins as a rule with marked redness and swelling of the gums. Most frequently the gums of the lower jaw, especially around the left incisor tooth, are first affected. The gums are unusually soft, and bleed readily on the slightest touch. Eventually they are transformed into a necrotic brownish or blackish mass, which on microscopic examination is found to contain round cells, red blood-corpuscles, at times algæ and infusoria, but especially innumerable bacteria. At times the gums are so completely destroyed that the teeth are loosened, and can be removed with the fingers without difficulty and without pain. The disease of the gums appears only in such situations where there are teeth. If any teeth are absent, the gums in those situations escape. Edentulous infants are not attacked by ulcerative stomatitis.

The patients complain of **pain** in the mouth, which is increased especially on the ingestion of food. The **secretion of saliva** is increased by reflex irritation. The saliva flows from the mouth, is often streaked with blood, and gives off a disagreeable odor. The skin of the chin is frequently reddened in streaks by the saliva and slightly inflamed. The sleep is often disturbed in consequence of the excessive flow of saliva, which may enter the larynx or be discharged upon the pillow. Patients usually exhale a horribly offensive **odor from the mouth,** which permeates the air of the room within a short time, and becomes evident even to the patient, causing a distaste for food and drink. Usually adjacent **lymph-glands,** particularly the submaxillary, undergo inflammatory swelling. The patients not rarely acquire within a remarkably short time a grayish, almost **cachectic appearance,** and they suffer from unusual languor. The **bodily temperature** generally remains unaltered.

The disease almost always terminates in recovery, although a patient under my observation died in consequence of septicemia. A distinction has been made between *acute* and *chronic ulcerative stomatitis*, accordingly as the disease terminates within one or two weeks or after the lapse of several months. In the absence of sufficient care in treatment *adhesions* may form between opposed ulcerated surfaces—as, for instance, between the mucous membrane of the cheek and the border of the tongue—and which often can be removed only with difficulty by operative means. At times the necrosis extends to the *lower jaw.*

Etiology.—A distinction should be made between *infectious ulcer* [illegible] *titis.* The *infectious* variety often occurs endemically, as, for instance, in barracks, jails, orphan-asylums, boarding-schools. Over-crowded, poorly ventilated, and damp dwellings favor its development, as do likewise deficiency of food and neglect in the care of the mouth. The conditions named render it explicable that the disease has been encountered almost exclusively among the poorer classes. Ulcerative stomatitis is observed most commonly in *children*. Anemic, rachitic, scrofulous, and otherwise debilitated children display a marked predisposition to the disease. The disorder occurs most commonly in the *summer months*.

Specific bacteria are as yet unknown in the etiology of ulcerative stomatitis. The Staphylococcus pyogenes aureus has been obtained from the necrotic tissue.

Toxic ulcerative stomatitis may be induced by mercurial preparations, either because these have been employed in excessive doses or because the individuals are unduly susceptible to the action of mercury.

Treatment.—**Causal treatment** will require the withdrawal of mercurial preparations, the provision of healthy dwellings, and food sufficient in amount and variety. **Symptomatically,** irrigation of the mouth after each meal with potassium chlorate (5.0 : 200) may be recommended. The food should consist especially of milk, weak wine, beer, broth, cocoa, coffee with milk and eggs. Hot and solid articles of food should be avoided on account of the pain in the mouth.

APHTHOUS STOMATITIS.

Symptoms, Anatomic Alterations, Diagnosis, and Prognosis.—Aphthæ appear as **yellowish or whitish deposits** upon the mucous membrane of the mouth, which are surrounded by a **red hyperemic zone,** and in contradistinction from masses of casein or of thrush cannot be detached with the finger. They occur with especial constancy upon the anterior aspect of the tongue, and upon the line of reflection from the gums to the mucous membrane of the lips. Their average size is between that of a lentil and that of a pea. They are at times roundish in shape, but by coalescence they may acquire an irregular shape. Their number is subject to wide variations.

The patients complain of a sense of **burning,** and even of pain in the mouth. Infants, therefore, often suddenly cease nursing, or altogether refuse nourishment. There is usually **increased secretion of saliva** from reflex irritation. The **general condition,** as a rule, exhibits no alteration. In the course of a few days the spots disappear, becoming smaller and smaller by absorption or by curling up at the margins, then becoming detached and exfoliated, and leaving a shallow ulcer, which within a short time becomes covered with epithelium, and heals without loss of structure. The disorder usually lasts from one to two weeks; at times, in consequence of repeated relapses, somewhat longer. It is unattended with danger. At times there may be **retention of saliva** in the salivary glands if aphthæ develop at the orifice of a salivary duct and cause obstruction. Exceptionally **acute nephritis** has been observed in the sequence of aphthous stomatitis.

Microscopic examination of the spots discloses the presence of fibrin admixed with a small number of round cells and bacteria (Staphylococcus pyogenes albus and aureus, the pneumococcus of Friedländer, the diplococcus of Stooss). It is assumed that aphthous stomatitis is an infectious disease, and that bacteria cause coagulation-necrosis of the epithelial cells of the buccal mucous membrane, so that fibrinous coagulation can take place in the exudate. The deeper layers of the mucous membrane are not involved in the inflammatory process.

Etiology.—Like most varieties of inflammation of the mouth, aphthous stomatitis occurs with especial frequency in *children*, particularly anemic, rachitic, and scrofulous children. Any *irritation of the buccal mucous membrane* may be followed by the development of aphthous stomatitis. In infants it is observed in association with the **irruption of the teeth**, with **uncleanliness of the mouth**, and with **irritation of the buccal mucous membrane from improper cleansing of the mouth.** Aphthous stomatitis may be associated with the most varied **diseases of the mouth** (catarrhal stomatitis, ulcerative stomatitis, thrush). It may result also from the irritation excited by **sharp projections of teeth.** In women the disease sometimes appears at the **menstrual period** or during **pregnancy** or **lactation.** At times it appears in the course of **infectious diseases** (typhoid fever, pneumonia, pulmonary tuberculosis, diphtheria). Occasionally aphthous stomatitis occurs *epidemically*, particularly in the summer months, and instances of contagion may also be observed.

Treatment.—In the first place, all obvious causes should be removed—**causal treatment.** In addition the mouth should be rinsed after each meal with **potassium chlorate** (5.0 : 200) or, in the case of small children, brushed or carefully swabbed.

THRUSH (STOMATITIS OIDICA).

Symptoms, Anatomic Alterations, Diagnosis, and Prognosis.—Thrush is attended with the formation of **whitish or yellowish deposits** upon the mucous membrane of the mouth, which can be **readily** detached with the finger or a hard object. Microscopic **examination** discloses the presence of a readily recognized **bacterium.** The ***thrush-fungus***, *Oidium albicans*, belonging to the **budding fungi, forms** in part septate thallus-filaments, in part **roundish or elongated** spores (Fig. 25). The tip and margins of **the tongue are as a rule** attacked first, although the proliferation in those with gastric disorders. *Men* are most commonly attacked, as may be readily understood from the nature of the causative factors. Often the disorder gives rise to no symptoms and is discovered purely by accident; in other instances complaint is made of soreness, burning, or pain in the mouth and of impairment of taste. Frequently the patients exhibit a hypochondriacal tendency, and are harassed by a fear that they are suffering from an incurable disease of the stomach or from syphilis.

The **prognosis,** however, should be cautious, as epithelioma has not rarely been observed to arise from leukoplakia.

The **treatment** consists, first, in the removal of causative influences. In cases of syphilis iodin should be administered internally and applied topically. Sufferers from gastric disease at times obtain relief, and are even cured, by courses of treatment at Carlsbad or Vichy. Among **medicaments** arsenic has been employed:

℞ Solution of potassium arsenite,
Bitter-almond water, each, 5.0 (75 minims).—M.
Dose: 10 drops thrice daily after meals.

In addition cauterization with lactic acid or chromic acid, topical applications of mercuric chlorid, argentic nitrate, tincture of iodin, or papayotin, and the galvanocautery have been recommended.

1. *Geographical tongue* is the name that has been given to an irregularly shaped collection of often confluent *red spots upon the tongue* lying below the level of the unchanged adjacent surface. Microscopic examination discloses in these situations desquamation of the uppermost layer of epithelial cells, round-cell accumulation among the remaining epithelial cells and in the papillary body, and a coagulated albuminous exudate in place of the desquamated epithelium. The disease either has been unattended with symptoms or has caused a sense of burning in the mouth. It has proved most obstinate and chronic, although the spots varied in shape from day to day. The disease attacks *children* most commonly, particularly anemic, scrofulous, and rachitic children.

2. *Black, hairy tongue* is characterized by a blackish discoloration of the tongue. Frequently the tongue presents also a felt-like roughness and multiple nodules. The condition is dependent especially upon hyperkeratosis of the epithelial cells of the filiform papillæ, the epithelial cells containing black and brownish pigment-granules. Such fungous formations as may be found are accidental contaminations that have nothing to do with the black pigmentations. The condition, which is often unattended with symptoms, or at most gives rise to a sense of burning in the mouth, occurs especially in nervous, debilitated patients and those suffering from gastric disorders, and it can only be corrected by mechanical removal and cauterization of the hyperplastic epithelial structures.

PTYALISM (SALIVATION).

Symptoms, Diagnosis, and Prognosis.—Ptyalism consists in **increased secretion of saliva,** so that either the patients are compelled to swallow the saliva frequently or the mouth is kept open and the saliva escapes, and must be received into a pocket-handkerchief or a receptacle of some kind. Often the integument of the chin is irritated by the discharge, and it is actively reddened, and slightly inflamed.

It should be noted that the escape of saliva is not a result of *paralysis of swallowing* (bulbar palsy). Earlier physicians designated this condition *false ptyalism.*

The collected saliva usually appears turbid, as it contains epithelial cells and remnants of food. Occasionally it yields an acid reaction, and at times potassium rhodanate and ptyalin are absent. The patients suffer most from the distress due to efforts at swallowing or in consequence of disturbed sleep. Some complain of a drawing sensation in the region of the parotid glands, and even of increased warmth and tension in the same situation. The amount of urine may be diminished. In ptyalism of paroxysmal occurrence I have observed the amounts of urine and saliva to vary reciprocally. The *duration of the disease* is variable. At times ptyalism is a disorder of a few hours or weeks, while at other times it may extend over years. In cases of chronic ptyalism progressive pallor and emaciation develop, because the large amounts of saliva swallowed interfere with gastric digestion. Ptyalism cannot be considered a disease directly dangerous to life, but the likelihood of cure depends upon the curability of the causative disorder.

Etiology.—Salivation results most frequently from **reflex irritation of the salivary nerves**; much less commonly there is **irritation of the salivary centers of the cerebrum or the medulla oblongata,** as, for instance, in consequence of central hemorrhage, softening, or new-growth. Ptyalism occurs also in the insane and the hysterical. The influence of the cerebrum upon the secretion of saliva is recognizable from the fact that the thought or the seeing of food causes an accumulation of water in the mouth, thus an increased secretion of saliva. In consequence of reflex irritation ptyalism occurs in association with most **diseases of the mouth and teeth.** The **irruption of the teeth** also is usually attended with increase in the secretion of saliva. Ptyalism often occurs in connection with **diseases of the stomach and bowel,** as, for instance, chronic **gastric catarrh** and the presence of tapeworm in the intestine. **Diseases of the liver, kidneys, and pancreas** also are not rarely attended with ptyalism. Frequently the reflex irritation emanates from the **sexual organs.** It is well known that ptyalism not rarely occurs during pregnancy. In some persons an excessive secretion of saliva occurs before and during coitus. **Sensory irritation** is at times attended with ptyalism, as, for instance, tickling of the skin or the hearing of shrill notes. Occasionally **inflammation of the salivary glands** themselves may be followed by ptyalism, but the opposite condition may likewise occur. At in those with gastric disorders. *Men* are most commonly attacked, as may be readily understood from the nature of the causative factors. Often the disorder gives rise to no symptoms and is discovered purely by accident; in other instances complaint is made of soreness, burning, or pain in the mouth and of impairment of taste. Frequently the patients exhibit a hypochondriacal tendency, and are harassed by a fear that they are suffering from an incurable disease of the stomach or from syphilis.

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Salivation may be excited intentionally by means of various medicaments, particularly *jaborandi-leaves* and their alkaloid, *pilocarpin hydrochlorate.*

Treatment.—In order to suppress systematically an increased secretion of saliva there is but one trustworthy remedy, namely, **atropin sulphate**, which may be injected subcutaneously or be administered internally:

℞ Atropin sulphate, 0.01 ($\frac{1}{6}$ grain);
Distilled water, 10.0 ($2\frac{1}{2}$ fluidrams).—M.
Dose: From 0.25 to 0.5 (4 to 8 minims), subcutaneously once or twice daily.

Or,

℞ Atropin sulphate, 0.005 ($\frac{1}{12}$ grain);
Powdered althea-root, sufficient to make 10 pills.
Dose: From 1 to 3 pills daily.

In addition **causal treatment** should be pursued, and the causes of the salivation should be removed.

An uncommon disease is *fibrinous sialodochitis.* This affection is attended with the formation of a fibrinous exudate and of coagula in the excretory ducts of a number of salivary glands, so that the discharge of saliva is prevented, and a sense of tension and swelling in the neighborhood of the affected glands develop. At times the disease occurs paroxysmally. Cure can be effected only by mechanical means, attempts being made to expel the coagula by pressure.

II. DISEASES OF THE PHARYNX AND THE SOFT PALATE.

CATARRH OF THE PHARYNX AND THE SOFT PALATE (CATARRHAL PHARYNGITIS AND ANGINA).

Etiology.—Inflammatory processes in the soft palate are [illegible] a sense of constriction [illegible]. A distinction should be made [illegible] of the *pharynx and soft* [illegible] adjacent infections, chemic, thermic [illegible] mechanical irritation, stasis, and inflammatory [illegible]

[illegible] acute or chronic course, and is [illegible]. When primary it [illegible], and when secondary [illegible] diseases among which may be mentioned scarlet fever, measles, syphilis, typhoid fever, erysipelas, and small-pox. Primary infectious inflammation of the pharynx and the soft palate was formerly attributed to exposure to cold, and it was therefore designated rheumatic (refrigeratory), but at present the tendency is correctly to believe that the inflammation is not excited by the cold alone, but that this, by causing changes in the circulation, diminishes the resistance of the tissues to the invasion of low forms of organisms, and thereby favors infection. Recent bacteriologic investigations have shown that *various bacteria* are capable of exciting inflammation of the soft palate; for instance, the Streptococcus pyogenes, the Staphylococcus pyogenes, the pneumoniacocci of Fränkel, the pneumococci of Friedländer, and others. Experience has shown that the tendency to infectious angina is most irregularly distributed. Anemic, debilitated, and delicate persons as well as rachitic and scrofulous individuals, are attacked by infectious angina with especial frequency. *Chronic hypertrophy of the tonsils* likewise favors the development of the disease. The affection occurs most frequently in *childhood*, and it is recognized that those who have recovered from an attack of infectious angina often suffer several times during the remainder of life from the same disease. Not rarely it occurs in epidemic distribution, or in institutions with many inmates endemics appear from time to time. Meteorologic influences are of importance in this connection, as the disease occurs with especial frequency and is widely distributed with sudden changes in the weather.

The *chemic irritants* that give rise to angina include, in addition to actual caustics (acids, alkalies, mercuric chlorid, argentic nitrate), also indulgence in alcohol and tobacco. Alcoholics and smokers of tobacco suffer mostly from chronic angina and pharyngitis, because in them the injurious influences are constantly operative. Certain medicaments taken internally also may give rise to inflammation of the soft palate and the pharynx, particularly potassium iodid and mercury.

Among *thermic irritants* hot articles of food are especially to be mentioned. Inhalation of hot vapors occurs much less commonly.

In consequence of *traumatic or mechanical irritation* angina and pharyngitis occur with especial frequency in conjunction with *inhalation of dust*, and it should therefore not excite surprise that those engaged in certain occupations (millers, furriers, stonecutters, hatmakers, bakers, etc.) frequently suffer from chronic angina and pharyngitis. Continued and loud speaking also is capable of exciting the disease, and it is therefore frequently encountered in speakers, singers, actors, teachers, officers, and waiters.

Hypostatic catarrh of the soft palate and the pharynx usually pursues a chronic course, and occurs most commonly in the sequence of chronic disease of the heart or of the respiratory organs.

Catarrhal pharyngitis and angina caused by *extension from adja-*

cent disease develops in conjunction with inflammation of the nasal, laryngeal, and gastric mucous membrane.

Symptoms, Anatomic Alterations, Diagnosis, and Prognosis.—The symptoms of *acute catarrh of the soft palate and the pharynx* occur in purest form in cases of primary infection. The patients either are suddenly seized with **fever**, which may have been preceded by a chill or slight shivering, or general malaise and languor may occur for several hours or days as prodromes. The fever may subside suddenly within twenty-four hours, often with perspiration, but it not rarely persists for several days. The patients at times present no other symptoms, and the affection may be recognized accidentally on inspection of the oropharyngeal cavity, resort to which may possibly be had because no explanation for the fever is obvious. Such conditions occur with especial frequency in children, in whom so-called ephemeral fever is often nothing more than the manifestation of an acute angina. **Labial herpes** often appears. I have also not seldom observed **enlargement of the spleen.**

As a rule, the *subjective symptoms* naturally direct attention to the diseased parts. The patients complain of **severe pain in swallowing and in speaking** at the junction of the buccal and pharyngeal cavities, where also a sense of constriction is appreciable. This is induced in part by the inflammatory swelling of the diseased structures, partly also by the increased sensitiveness of the inflamed tissues. The pain is all the more noteworthy because the patients are compelled to swallow frequently in consequence of increased secretion of mucus. Yawning also induces pain, as the inflamed tissues are stretched in consequence of this act. Often the **nasal speech** is conspicuous, and it appears when the tonsils are greatly swollen and obstruct the nasopharyngeal space. Under such circumstances the mouth is generally kept open, in order that air may enter the larynx—**mouth-breathing**. The patients often hold the **head stiffly**, and avoid rotatory and nodding movements, because pain in the inframaxillary region and in that of the soft palate is thereby induced. On palpation of the inframaxillary region painful, hard, roundish structures are found on one or both sides in the neighborhood of the angle of the jaw—**inflamed and enlarged submaxillary lymphatic glands**—representing the consequences of secondary infection. If the mouth be opened widely and the dorsum of the tongue is depressed with a broad, hard body—for instance, the handle of a firm spoon—the changes in the inflamed structures can be well studied, and with greater accuracy than on the cadaver. These appear in three forms, which may be designated as [illegible], [illegible], and [illegible] inflammation.

In cases of [illegible] the affected areas are characterized by [illegible] redness and swelling and increased secretion of mucus. At times [illegible] of the mucous membrane appear as small, enlarged, almost transparent nodules, partly because they contain retained secretion and partly because inflammatory cellular hyperplasia has taken place in them. In consequence of rupture superficial follicular ulcers may develop. Here and there, also, superficial ulceration of the mucous membrane may occur in places where the epithelium has been desquamated. The alterations may be confined to circumscribed areas or they may involve the entire mucous membrane of the soft palate and the pharynx, so that a distinction must be made between *circumscribed* and *diffuse angina and pharyngitis.*

Lacunar angina is attended with the formation of yellowish or yellowish-gray deposits in the lacunæ of the tonsils, so that the latter are often strewn with nodules averaging about the size of a pin-head. Often each nodule is surrounded by a red hyperemic zone. Not rarely the deposits extend gradually beyond the limits of the individual lacunæ and coalesce, so that yellowish or grayish-green smeary deposits are visible upon the tonsils and, not rarely, also upon the uvula and the arch of the palate. On microscopic examination these are found to consist of round cells, fat-globules, needles of fatty acids, and cholesterin-plates, but especially of bacteria (leptothrix-filaments, Streptococcus pyogenes, Staphylococcus pyogenes, pneumoniacocci, etc.). The same anatomic alterations that occur in superficial and lacunar catarrh occur also in *pharyngeal diphtheria.* The differential diagnosis can be made only by means of bacteriologic examination, for in all diphtheric diseases diphtheria-bacilli are found in the inflammatory products.

Parenchymatous inflammation, also designated *phlegmonous angina*, is characterized by the formation of a circumscribed collection of pus, thus an abscess. It is usually attended with intense pain. The inflamed area is conspicuous for its great redness and prominence. The abscess often ruptures into the oropharyngeal cavity in the course of a few days, when the pain at once diminishes and the swelling is reduced in size. Often the inflammation disappears in the course of a few days. Most frequently the accumulation of pus takes place in the periglandular connective tissue about the tonsil—so-called tonsillar abscess. Disagreeable complications arise at times in consequence of rupture of the abscess and entrance of the pus into the larynx during sleep, with cough and danger of suffocation; or in consequence of rupture of the abscess externally, with dangerous erosions of large blood-vessels in the neck, or burrowing downward of the pus, or rupture through the skin of the neck.

Most cases of acute angina and pharyngitis terminate favorably within a few days. Among *complications* may be mentioned *albuminuria*, which often disappears from one day to another, *acute nephritis, paralysis of the muscles of the palate*, and *septic alterations (multiple, painful articular swelling* and *endocarditis*). The nviction has but recently been arrived at that the tonsils fre-uently constitute the portal of entry for bacteria into the body, ven when those structures themselves remain unchanged.

Chronic inflammation of the soft palate and the pharynx is unattended with fever, elevation of temperature taking place only when acute exacerbations occur. *Superficial, lacunar,* and *parenchymatous forms of chronic inflammation* also may be distinguished.

In *superficial chronic angina and pharyngitis* the inflamed parts are notable for their brownish-red or grayish-red color and the abundant secretion of viscid mucus. Frequently a number of the vessels of the mucous membrane are tortuous and exhibit varicose dilatation. Bleeding may take place from these and give rise to hemorrhagic expectoration, so that many persons suffering from chronic pharyngitis believe themselves to be suffering from pulmonary tuberculosis. Owing to the presence of abundant secretion the mucous membrane frequently appears glazed. At times the mucus dries during the night into grayish-green mussel-like crusts, which in the morning occasion a feeling as if a foreign body were present, and often can be expelled only with difficulty after protracted hawking and struggling. Often the lymph-follicles of the mucous membrane undergo hypertrophy, and also new lymph-follicles form, so that the posterior wall of the pharynx especially is strewn with small gray nodules—*granular pharyngitis.* The patients complain of a feeling as from the presence of a foreign body, even of pain and soreness in the pharyngeal cavity, while an abundance of viscid mucus gives rise to increased frequency in the act of swallowing, and often to retching and nausea. The symptoms mentioned usually become apparent in speaking and singing, particularly also in the morning if during the night there has been opportunity for the mucus to collect and become adherent in the pharyngeal cavity, and it is a common observation that such patients begin the day with a rather protracted period of hawking and retching, which subside only with the completion of the toilet of the pharynx. *Impairment of hearing* and *tinnitus aurium* occur not rarely as *complications,* either because the buccal orifice of the Eustachian tube becomes occluded by swelling of the mucous membrane of the pharynx or because the catarrhal condition itself has extended to the mucous membrane of the tube, and at times even to the tympanic cavity. Although the affection is not a fatal one, it is nevertheless an extremely obstinate and troublesome one, as it often renders the pursuit of the patient's occupation most difficult, and at times even impossible.

In *chronic lacunar angina,* as in the acute form, yellowish or grayish smeary masses accumulate in individual lacunæ of the
ils consisting principally of bacteria, and especially of lepto-
When crushed these plugs emit a most offensive
is principally of a sense of tickling

or of the presence of a foreign body, and from time to time hawk up the accumulated plugs. At times these undergo calcification, and form tonsillar calculi, and are from time to time expelled by hawking. They may possibly be mistaken by the patient for calcified tubercles from the lungs. The disagreeable odor may be a source of distress to the patient.

Parenchymatous angina and pharyngitis are attended with inflammatory hypertrophy of the diseased structures. If the tonsils are involved in the inflammation, they may attain double their normal size, and they not rarely project into the entrance of the pharynx to such a degree as to come in contact in the middle line. In consequence *speech* suffers and acquires a nasal character; besides, the patients are consequently compelled to breathe through the mouth, and they snore during sleep. Finally the orifice of the Eustachian tube becomes obstructed, and impairment of hearing and tinnitus aurium result. It should not be forgotten that inflamed and hypertrophied tonsils manifest a tendency to acute inflammation, and frequently are the seat of acute recurrent disease. In some cases the uvula undergoes hypertrophy, and its tip may come to lie upon the dorsum of the tongue. The patients then complain frequently of retching and nausea.

At times chronically inflamed pharyngeal mucous membrane presents a pale, atrophic, and attenuated appearance—*chronic atrophic pharyngitis.* Frequently similar alterations are at the same time present upon the nasal mucous membrane.

Of the chronic inflammatory conditions of the soft palate and the pharynx it may be said that none is dangerous to life, while all are distressing affections, curable with difficulty, particularly as they are often dependent upon the occupation or upon bad habits (smoking, alcohol), which the patients are unwilling to give up. Not rarely chronic angina and pharyngitis predispose to the occurrence of chronic catarrhal conditions of the nasal, laryngeal, tracheal, and bronchial mucous membrane. At times nervous complications also arise through reflex influences, as, for instance, bronchial asthma, and even epilepsy.

Treatment.—Patients suffering from *acute angina and pharyngitis* should remain in bed, and partake only of a liquid diet, preferably milk, and inhale every two or three hours a solution of potassium chlorate through the apparatus of Siegle. Gargles are less worthy of recommendation on account of the unavoidable irritation of the inflamed structures:

℞ Potassium chlorate, 5.0 : 200.
For inhalation (or gargling) every two or three hours.

If the mucous membrane is the seat of severe pain and great swelling, bits of ice may be sucked. The application about the neck of a long rubber bag filled with ice may also be recommended—a so-called ice-collar. If parenchymatous inflammation is complicated by the formation of an abscess, escape of the pus externally must be provided for by **incision.** *Chronic angina and pharyngitis* demand, in the first place, thorough consideration of the causative influences, **causal treatment,** and, for instance, excessive indulgence in alcohol and tobacco must be as rigidly as possible enjoined, and the patient should be advised to avoid loud and continuous speaking and the inhalation of dust. **Symptomatic treatment** consists principally in the local employment of gargles, topical applications, insufflations, cauterization, inhalations, and a sojourn at certain springs. At times surgical intervention becomes necessary. For *gargling* solutions of potassium chlorate, alum, sodium chlorid, sodium bicarbonate, and ammonium chlorid have been recommended—all in the proportion of 5.0 : 200. The therapeutic agent must be varied from time to time, as the mucous membrane readily becomes accustomed to the same remedy, and is then no longer susceptible to its influence. For *topical application* dilute tincture of iodin especially may be recommended, but it is to be withheld if a more acute inflammation results in consequence of the application. Immediately after the application the patient should gargle with water:

℞ Tincture of iodin,
Tincture of galls, each, 5.0 (75 minims).—M.
Use daily as an application with a brush.

Insufflations are made with either a glass tube or a quill, and tannic acid, alum, or iodoform may be employed for the purpose. *Caustics* (silver nitrate, copper sulphate, chromic acid, the galvanocautery) are especially applicable to granular pharyngitis. *Inhalations* may be practised with the aid of Siegle's apparatus, and the same medicaments are employed as have been named for gargling. Courses of treatment at *springs* often yield good results because the patient is for a time removed from the exciting causes. Especial repute in this connection is enjoyed by ***sulphur-springs*** (Aachen, Baden, Heustrich, Nenndorf, Schinznach, Stachelberg, Weilbach), *chlorid-springs* (Ems, Homburg, Kissingen, Reichenhall, Soden, Wiesbaden), and *iodin-springs* (Salzbrunn, Tölz, Adelheid Springs). Hypertrophied tonsils and elongated uvulas should be removed by surgical means. In order to avoid recurrences of inflammation of the soft palate and the pharynx certain prophylactic measures should be observed. These will vary in accordance with the nature of the exciting cause, and consist in part in intelligent hardening of the body and in part in protection against inhalation of dust and the like.

MYCOSIS PHARYNGIS LEPTOTHRICIA.

This disorder is attended with the formation of *yellowish spots,* often apparently encapsulated, which lie especially in the lacunæ of the tonsils, and on microscopic examination are found to consist principally of *leptothrix-filaments.* The disease either is unattended with symptoms, and is discovered accidentally, or it gives rise to the feeling of a foreign body and to efforts at retching. At times the fungous granulation extends to the dorsum of the tongue, especially to the circumvallate papillæ, and to the mucous membrane of the larynx and even of the trachea. The affection, which is in itself free from danger, is usually most obstinate. At times it has been observed to disappear spontaneously; at other times it seems to be dissipated by *smoking.* In addition, *gargling with mercuric chlorid* (1.0 : 2000), and *mechanical removal* of the deposit and its destruction by means of the *galvanocautery,* have been employed.

III. DISEASES OF THE ESOPHAGUS.

CARCINOMA OF THE ESOPHAGUS.

Anatomic Alterations.—Carcinoma of the esophagus is almost exclusively of *primary origin.* Only a few instances of secondary carcinoma, arising from dissemination of the germs of the disease from other organs previously the seat of carcinoma, are known. Most frequently carcinoma of the esophagus is *situated* just above the cardia, but somewhat less commonly it develops at the level of the bifurcation of the trachea, and least commonly it occurs at the beginning or in any other portion of the esophagus. It is rare for isolated carcinomata to be present in several portions of the esophagus. The new-growth begins as a small *insular* formation, and then forms upon a more or less extensive portion of the mucous membrane a roundish nodule projecting into the lumen of the esophagus. As a rule, it is gradually transformed into a **girdle-shaped** or **annular carcinoma,** as it encircles the entire circumference of the esophagus like a ring. The mucous membrane in the diseased area is thickened to the extent of several centimeters, and consists here of a whitish, medullary, friable tissue. This may be observed also in the muscular layer of the esophagus, in which extension within the connective-tissue septa takes place. The muscular layer undergoes thickening, and even the external surrounding connective tissue.

Histologically, carcinoma of the esophagus is always of the *pavement epithelial variety.* The morbidly proliferated, squamous epithelial cells are derived from the epithelial cells of the mucous membrane, but in part also from those of the glands of the mucous membrane.

As may be understood, *stenosis of the esophagus* occurs at the situation of the new-growth. Above the point of constriction dilatation of the esophagus takes place at times, but by no means constantly, while, below, the esophagus may be unusually narrow. The **tracheobronchial** glands adjacent to the esophagus are usually enlarged and the seat of carcinomatous infiltration. Less commonly similar alterations are present also in the cervical lymphatic glands. Like carcinoma elsewhere, that of the esophagus also exhibits a tendency to *constant growth* and to *disintegration*. The first quality is responsible for the fact that in the course of the disease the constriction of the esophagus increases, and that at times adjacent organs are compressed and impaired in function, as, for instance, the recurrent laryngeal nerve. As a result of disintegration of the carcinomatous tissue ulceration takes place, leaving an eroded and villous, and at times an offensively smelling, surface. The destruction may become so extensive that scarcely any carcinomatous tissue is visible, and the muscular layer of the esophagus is exposed as if dissected free. Formerly it was even erroneously supposed that the ulcer underwent cicatrization, and that the carcinoma was as a result capable of being entirely healed. The disintegration of the carcinomatous tissue further is attended with the danger that adjacent organs may be attacked, and *rupture of the esophagus* take place into them (pleura, lungs, trachea, mediastinum, aorta, pericardium). Persons dying of carcinoma of the esophagus present extreme emaciation, brown atrophy of the myocardium, and almost empty intestines.

Etiology.—Clinical observation has shown that carcinoma of the esophagus, like carcinoma of other organs, occurs *late in life* (after the fortieth year), and in *men* with twice the frequency that it occurs in women. Immediate causes are, as a rule, not elicitable, although I have observed the disease in numerous instances in **drinkers** and **smokers**, so that I would ascribe some etiologic influence to the irritation of the mucous membrane of the esophagus by alcohol and swallowed tobacco-juice. Some patients attribute the disorder to the entrance into the larynx of **hot articles of food** or **foreign bodies**. In a few instances carcinoma has been observed to develop from **ulcers** of the esophagus.

Symptoms.—The most constant, and frequently also the first, symptom of carcinoma of the esophagus consists in spontaneously developed and progressively increasing stenosis of the esophagus. In other instances other *suspicious symptoms* have appeared, first among which particularly pain and paralysis of the recurrent laryngeal nerve are to be mentioned. I have at times observed **pain** to precede by months the earliest difficulty in swallowing. The pain always remained confined to the same situation, and was at times referred to the vertebral column, and at other times behind the sternum. It frequently increased in intensity during the night and disturbed sleep. No resistance was found on introducing a bougie into the esophagus, but the same point in the esophagus remained constantly sensitive on contact with the instrument.

Paralysis of the recurrent laryngeal nerve as a result of carcinoma of the esophagus is due either to pressure or to proliferation of the new-growth in the trunk of the nerve. Usually it is the left recurrent nerve that is affected, but less commonly both are involved in the paralysis. Unilateral paralysis of the recurrent laryngeal nerve can be recognized only by laryngoscopic examination (cadaveric position of the paralyzed vocal band, page 91, Fig. 17); when both vocal bands are paralyzed disorders of speech, in cough, and in expulsion are also present. In some cases my attention was attracted entirely by **secondary carcinoma in other organs** (liver, cranium), and the carcinoma of the esophagus was overlooked because the patients failed to complain of either difficulty in swallowing or pain in the esophagus.

Progressive difficulty in swallowing, in consequence of increasing **stenosis of the esophagus**, begins as a rule gradually. Exceptionally, however, I have observed it to set in suddenly, and further after great mental and physical exertion and strain. The patients generally note at first that customary boluses of food will not pass beyond a given point in the esophagus, but threaten to remain impacted. The endeavor is made to avoid this circumstance by the swallowing of smaller boluses, but these after a time also encounter obstruction. Other expedients are attempted, such as more thorough chewing of the food and more complete insalivation of the food, together with rotatory movements of the head and neck, but these also fail after a time. Finally, the only resource left is to restrict the diet to fluids, because all solid substances are regurgitated in a macerated state shortly after ingestion, and excessive distention of the esophagus with solid material gives rise, by pressure partly upon the trachea and partly upon the adjacent vagus, to shortness of breath, a sense of anxiety, and palpitation of the heart. The ingestion of liquids, also, is attended with increased resistance, and thus the patient passes gradually into a state in which no food can be introduced into the stomach and the bowel through the mouth, and death from starvation cannot be long deferred. It is distinctive of the food regurgitated that, in contradistinction from the acid contents of the stomach, it yields a neutral reaction and not rarely has a fetid odor. Amylacea are partially transformed into sugar in the esophagus by the saliva, and the patients therefore often state that the regurgitated matters possess a sweetish taste. Further, repeated ***variations in the degree of stenosis of the esophagus*** occur in the course of the disease, partly because portions of the new-growth are broken off, and the lumen of the esophagus is thus increased, partly because at times ingested materials become impacted above the constriction.

The patients undergo progressive emaciation, and the skin acquires a grayish-yellow or earthy hue and a leathery and wrinkled appearance. Owing to their asthenia the patients are compelled to remain in bed, and frequently on assuming the erect posture they suffer, in consequence of cerebral anemia, from tinnitus aurium, obscurity of vision, palpitation of the heart, and syncope. At times delirium due to inanition occurs. The abdomen is generally scaphoid on account of the empty state of the intestines, and the bowels are obstinately constipated. The urine is voided in small amounts and contains much indican. The bodily temperature is often subnormal. Progressive apathy sets in, and finally gradual dissolution takes place. The *duration of the disease* is usually more than six and less than twenty-four months. Naturally, when carcinoma of the esophagus is suspected investigation for objective evidence of the presence of stenosis of the esophagus should never be omitted, and the only certain way of doing this consists in the *introduction of a bougie into the esophagus.*

The most serviceable bougies are the *English red bougies* with lateral fenestra. If it be desired to determine the degree of stenosis, bougies of varying size should be employed. It is important to examine microscopically with care the substances caught in the fenestra, as these sometimes consist of carcinomatous tissue, consideration being given especially to the abundant presence of pavement epithelial cells, with numerous nuclei, and to spherical laminated epithelial cells (carcinoma-nests or carcinoma-pearls). If these structures be not found, the examination with the bougie will disclose only the presence of stenosis of the esophagus, and the carcinomatous character of the affection must be determined from other circumstances. In order to learn the *seat of the disease* the simplest method consists in applying the bougie externally along the mouth and the vertebral column, and noting the position of the tip of the instrument. The observation can be made more precisely by measuring the bougie with a centimeter-measure and comparing the result with the average measurements of the esophagus. The following are the *dimensions of the esophagus:*

Entire length of the esophagus, 25 cm. (9.8 inches).

The distance from the edge of the teeth to the beginning of the esophagus, 15 cm. (5.9 inches).

Length of the cervical portion of the esophagus, 5 cm. (1.9 inches).

Length of the dorsal portion, 17 cm. (6.7 inches).

Length of the abdominal portion, 3 cm. (1.2 inches).

The point of intersection between the esophagus and the left bronchus cross is:

From the beginning of the esophagus, 8 cm. (3.1 inches).

From the edge of the teeth, 15 + 8 = 23 cm. (9 inches).

Should a bougie not be at hand, tentative observations should be made by *auscultation of the esophagus.* The stethoscope is applied along the left side of the vertebral column from the seventh cervical to the eleventh dorsal vertebra. If the patient swallows water when directed, there is heard in healthy persons throughout the entire course of the esophagus a peculiar sound like that which every one may hear when the ears are closed with the fingers and the act of swallowing is practised. In persons suffering from stenosis of the esophagus this swallowing sound is audible only down to the point of constriction, while below this it is wanting. The *swallowing sounds of the stomach* also are altered in the presence of constriction of the esophagus. In order to detect these the stethoscope is placed in the angle between the ensiform cartilage and the left costal margin while water is swallowed. In healthy persons two murmurs will be audible in from five to ten seconds, the first of which is designated the *injection-sound* and the second the *expression-sound.* These sounds are explained by the fact that the ingested fluid is usually forced through the esophagus into the stomach by the contraction of the muscles of the oropharyngeal cavity, and that then the remainder is forced into the stomach by contraction of the musculature of the esophagus. When the esophagus is constricted the injection-sound occurs after an unusually long interval, and often appears gurgling and prolonged, while the second sound is often wanting. For the detection of alterations in the esophagus, examination with mirrors—*esophagoscopy*—has been proposed, but this has not yet received general application, principally because the instruments thus far constructed are not sufficiently manipulable on the part of both physician and patient.

Carcinoma of the esophagus is not rarely attended with *complications,* which may give rise to serious consequences. *Paralysis of the recurrent laryngeal nerve* has already been considered. Bilateral recurrent paralysis renders easily possible the entrance of foreign bodies into the air-passages, with the development of *aspiration-pneumonia, abscess,* and *gangrene of the lungs.* Similar alterations in the lungs occur not rarely also without recurrent paralysis, because in the act of regurgitation individual masses of food readily enter the larynx and the deeper air-passages. In some instances I have observed symptoms of *paralysis of the sympathetic nerve* (contracted pupils, narrowing of the palpebral fissure, recession of the eyeball). Occasionally serious *hemorrhage* takes place, at times soon after the introduction of a bougie into the esophagus, and at other times spontaneously. In the latter event the bleeding may be due to disintegration of carcinomatous tissue and perforation of blood-vessels, or to erosion of adjacent large blood-vessels, as, for instance, the aorta. Under such circumstances death may take place rapidly from hemorrhage. Often *rupture of the esophagus into adjacent organs* takes place, most frequently into the left bronchus. The patients are then seized with severe cough after the ingestion of food, and expectorate portions thereof. Further, aspiration-pneumonia and abscess and gangrene of the lung soon develop, because a portion of the food enters the deeper air-passages. In other instances rupture takes place into the larynx, directly into the lungs, into the pleural cavity, or into the mediastinum. If air enters the mediastinal connective tissue from the esophagus, it generally extends upward beneath the connective tissue of the jugular fossa, with the development of subcutaneous emphysema (projection of the skin, crepitation on manipulation). I have frequently observed rupture of the esophagus preceded for several days by inexplicable febrile movement. At times carcinoma of the esophagus extends to the vertebral column, destroying the vertebræ, and giving rise to *spinal pressure-paralysis.* The complications include further *secondary* or *metastatic carcinoma in other organs*, as, for instance, in the liver, and these may dominate the clinical picture to such a degree that the primary growth in the esophagus will be overlooked.

Diagnosis.—The diagnosis of carcinoma of the esophagus is usually indirect. In a person at a susceptible age constriction of the esophagus is found, and its carcinomatous nature is decided upon because of its development without demonstrable cause, its gradual increase, its advance with progressive asthenia, and its continuance without appreciable alteration in organs contiguous to the esophagus. It is rare that portions of the new-growth are caught in the fenestra of the bougie that on microscopic examination can be definitely recognized as carcinomatous, or that portions of the growth are found in the regurgitated matters. Especial care should be taken to determine that the stenosis of the esophagus is not due to an **aneurysm of the aorta**, and in no instance should a bougie be introduced into the esophagus until aneurysm of the aorta has been excluded by the absence of abnormal dulness, vascular murmurs, and alteration in pulse. If this precaution be neglected, it may happen that an aneurysm of the aorta compressing the esophagus may be perforated by the tip of the bougie, and death result rapidly from hemorrhage.

With regard to *other varieties of stenosis of the esophagus* a few remarks of etiologic significance may be appropriate at this place. The symptoms and the treatment agree essentially with those of the stenosis due to carcinoma. In accordance with the seat of the constriction a distinction is made between *intra-esophageal*, *parietal*, and *extra-esophageal stenosis of the esophagus*. *Intra-esophageal stenosis* may be due to the lodgment of *articles of food* and *foreign bodies* and *proliferated thrush-masses*. *Parietal stenosis of the esophagus* may result from *cicatrices* in the sequence of burns, corrosion, tuberculous, diphtheric, peptic, and syphilitic ulcers, and variola. It occurs in association with *new-growths* (carcinoma, polyps, gummatous nodules) and *abscesses*. *Pulsion-diverticula* when filled give rise to constriction of the esophagus. Among other causes for constriction of the esophagus that have been mentioned are *congenital stenosis*, *hypertrophy*, and *spasm of the musculature of the esophagus*. *Extra-esophageal stenosis* may also be designated *compression-stenosis of the esophagus*. The following examples may be mentioned: Aneurysm of the aorta, pericarditis, pleurisy, new-growths of the lung, the mediastinum, the pleura, the pericardium, the vertebral column, the thyroid gland (struma), ossification and thickening of the cricoid cartilage, burrowing abscesses of the vertebral column, luxation of the hyoid bone.

Prognosis.—The prognosis of carcinoma of the esophagus, as of carcinoma elsewhere, is unfavorable, and a fatal issue is unavoidable.

Treatment.—Internal remedies for the cure of carcinoma are not known, and the only reliable method of treatment consists in removal of the growth with the knife. As a matter of fact, **resection of the carcinomatous portion of the esophagus** has been attempted, but the operation naturally can only be undertaken when the carcinoma is situated in the cervical portion of the esophagus and is thus accessible to the knife—and this is uncommon. In the majority of cases **symptomatic treatment** alone is possible, and the indications are by means of nutritious food to maintain the strength and to prolong life as well as possible, and to preserve the permeability of the esophagus. The most suitable diet consists in liquids and soft food. Among these may be mentioned milk with somatose, broths, milk with tea, milk with chocolate, milk with cocoa, eggs, wine, beer, rice, oatmeal-gruel prepared with milk, beef-tea, scraped ham or tenderloin, tender meat-sausage, and chopped meat. Food should be taken every hour, but slowly and not too much at a time. In order to maintain the patulousness of the esophagus for as long a time as possible **systematic employment of the bougie** should be practised daily or at intervals of several days in accordance with existing conditions. Even if the sound cannot be passed through the constricted portion of the esophagus, deglutition will be rendered materially easier or again possible. Attempts have been made to introduce permanent catheters through the constricted portion of the esophagus and permit them to remain, but objection has been raised to this procedure from various sources that it favors disintegration of the tumor. If the stricture of the esophagus has become impassable, two methods only remain of introducing food into the body—either **gastrostomy** or **nutrient enemata.** Gastrostomy has heretofore not been attended with brilliant results, perhaps because it has generally been undertaken too late; nor can life be prolonged for any considerable length of time by means of nutrient enemata, because it is impossible to secure absorption of a sufficient amount of nutrient material from the mucous membrane of the large intestine.

The following formula may be recommended as a nutrient enema: 150 c.c. of lukewarm milk, 150 c.c. of lukewarm, slightly salted meat-broth, two eggs, 3 teaspoonfuls of somatose, 2 tablespoonfuls of starch-flour, 1 teaspoonful of sugar, and 10 drops of tincture of opium. By means of a Hegar funnel a mixture of this sort at the temperature of the body is permitted to flow into the rectum thrice daily—in the morning three hours after having evacuated the contents of the rectum by means of cold water. Great difficulty arises from the circumstance that the patients, by reason of their weakness, are unable to retain the enemata, or that, in spite of all care, the mucous membrane of the rectum soon becomes irritable, and in consequence retention of the enemata is impossible.

DIVERTICULA OF THE ESOPHAGUS.

A diverticulum of the esophagus consists in *circumscribed dilatation* of this tube. A distinction is made between *pulsion-diverticula* and *traction-diverticula*, accordingly as the dilating force is applied from within outward or traction is exercised from without.

Pulsion-diverticula are almost always situated at the junction of the pharynx with the esophagus, and are rather a part of the former. Most commonly they arise in the middle line, bulging outward from this situation in one or both directions. Under such conditions they may attain the size of a child's head. On anatomic examination they are frequently found to be unsupplied with a muscular layer, so that it has been correctly assumed that the tendency to the development of a pulsion-diverticulum is congenital, and is attributable to deficient development of the muscular layer, and therefore to diminished resistance on the part of the wall of the esophagus. Other observers are of the opinion that pulsion-diverticula result from incomplete internal cervical fistulæ in the region of the first branchial cleft. Persons with pulsion-diverticula of the esophagus frequently state that, after eating, a *swelling* forms on the left side of the neck, which gradually increases in size with the further ingestion of food. Pressure and manipulation may cause disappearance of the swelling, while the patients are conscious of the entrance of food into the esophagus. At the same time loud gurgling *murmurs* are not rarely generated. The diverticulum has a tendency to intercept swallowed articles of food, and during the process of eating to have its orifice directed more and more into the lumen of the esophagus. Often *regurgitation of the food* takes place some time after its ingestion. The ejected matters appear macerated, and often emit a fetid odor. On *introducing a bougie into the esophagus* it will be observed that at times the instrument passes readily into the esophagus, while at other times it is obstructed if its extremity enters the diverticulum. It may also happen that if two bougies are introduced into the esophagus the one will enter the diverticulum, while the other passes into the stomach without obstruction. By *distention* with the aid of a teaspoonful each of sodium bicarbonate and tartaric acid, given separately in a wineglassful of water, it may be possible to induce a prominence in the neck, which on percussion yields a tympanitic note. A pulsion-diverticulum is attended with the danger of progressive emaciation and of *starvation*, for in addition to the interception of food a distended pulsion-diverticulum, by projecting into the esophagus, prevents the passage of food to the stomach. Pulsion-diverticula have recently been excised successfully; otherwise the only remedy would consist in systematic nutrition by means of the stomach-tube. Pulsion-diverticula occur *most commonly in men*. The earliest symptoms usually do not appear until the fortieth year of life, and they are often attributed to swallowed foreign bodies, injuries of the cervical region, and constricting articles of clothing. Ossification and thickening of the cricoid cartilage occurring late in life appear also to be of causative influence.

Traction-diverticula of the esophagus are not recognizable during life on account of their smallness. They are most frequently situated at the level of the bifurcation of the trachea, for they are principally dependent upon inflammatory alterations in the tracheobronchial lymph-glands. In consequence of periadenitis adhesions readily form between the lymphatic glands and the esophagus, and if subsequently contraction and diminution in size take place in the inflamed lymphatic glands the wall of the esophagus is drawn outward in the form of a small, acute funnel. Often the glandular structure will be visible at the apex of the funnel as a dark body. The danger from traction-diverticula resides in their tendency to *rupture*, and this may occur spontaneously or be caused by incarcerated, hard particles of food. Rupture may take place into the air-passages, when the entrance of food gives rise, in addition to distressing irritative cough, also to inflammation, suppuration, and gangrene of the lungs; or into the mediastinal connective tissue, where it gives rise to putrefaction or to accumulation of air, or to both; or into the pleura, the pericardium, or the aorta, with the development respectively of pleurisy or pericarditis, or the occurrence of death from hemorrhage within a short time.

DILATATION OF THE ESOPHAGUS.

Dilatation of the entire esophagus may be *congenital* or *acquired*, and in the latter event some of the causes mentioned include frequent vomiting, excessive ingestion of hot water, impaction of hot food, chronic catarrh of the esophagus, a blow upon the chest, and the carrying of heavy weights. The dilatation of the esophagus is at times spindle-shaped and at other times more uniform. In the first instance, the greatest degree of dilatation usually corresponds with the mid-dorsal region. The dilated esophagus is at times capable of enclosing a man's arm, but it is also considerably increased in length, and at times makes numerous lateral turns in its course. Dilatation of the esophagus may be recognized from the fact that considerable lateral movement can be made with a sound that has been introduced. If, on the other hand, the esophagus makes lateral turns, the introduction of the bougie may be attended with considerable difficulty. Under such conditions the patients also complain of difficulty in swallowing and often regurgitate ingested matter, and they may slowly undergo great emaciation and die in consequence of inanition. Experience has shown that difficulty in swallowing generally does not occur until after the twentieth year of life.

Partial congenital dilatation of the esophagus constitutes the so-called *ante-stomach*, which consists in a dilatation of the portion of the esophagus that is situated below the diaphragm. The condition is without significance, and has nothing to do with rumination, as was formerly supposed.

Secondary dilatation of the esophagus takes place above constrictions of the esophagus, but it is by no means a necessary consequence of such conditions. The dilatation becomes the greater the greater the amount of food ingested and the longer it remains above the constriction.

CATARRHAL ESOPHAGITIS.

Etiology.—Catarrh of the esophagus is of only subordinate clinical importance. It develops, like catarrhal conditions of other mucous membranes, in consequence of the action of **mechanical** (hard articles of food, swallowed foreign bodies, careless introduction of the sound), **toxic** (the swallowing of acids, alkalies, and other corrosive substances), and **thermic irritants** (hot articles of food, abuse of alcohol and of tobacco). **Infectious influences** probably play a part in the inflammatory processes that develop in the course of certain infectious diseases (small-pox, measles, scarlet fever, typhoid fever, erysipelas). At times inflammation of the mucous membrane of the esophagus arises by *extension* from catarrhal conditions of the stomach, the pharynx, the larynx, the bronchi, from mediastinitis, pericarditis, and pleurisy. A special variety is constituted by the *hypostatic catarrh* that is observed in connection with chronic cardiac and respiratory diseases. In accordance with the course of the disease a distinction has been made between *acute* and *chronic*, and, in accordance with its distribution, between *diffuse* and *circumscribed catarrh of the esophagus*.

Anatomic Alterations.—*Acute catarrh of the esophagus* is characterized less by unusual engorgement with blood and redness of the mucous membrane, than by marked detachment and desquamation of the epithelium. The follicles of the mucous membrane, in consequence of accumulation of secretion, often appear as small, clear and transparent nodules, which not rarely are arranged upon the summit of the folds of mucous membrane like strings of pearls. If follicles rupture, superficial **follicular ulcers** result. In addition, if excessive desquamation of epithelial cells takes place **epithelial ulcers** form.

Chronic catarrh of the esophagus is attended with brownish-red discoloration of the mucous membrane and thickening of the epithelium. The muscular layer, also, may undergo inflammatory thickening. At times dilatation of the esophagus takes place. The abundant formation and accumulation of mucus further constitute a striking feature.

Symptoms, Diagnosis, and Prognosis.—The morbid manifestations are in part indefinite in character, including deep-seated pain in the course of the vertebral column, or between the scapulæ, pain in swallowing—**odynphagia**, at times **difficulty in**

swallowing, and even **regurgitation**. In cases of acute catarrh it has frequently been observed that plugs of desquamated epithelial cells from the mucous membrane were ejected. The *introduction of a bougie* into the esophagus should if possible be avoided, in order that no irritation be excited. It is noteworthy that an abundance of mucus, at times blood-tinged, frequently adheres to the sound. The *duration* of the disease and the *prognosis* depend upon the fact whether the cause can be removed or not.

Treatment.—In cases of *acute catarrh of the esophagus* only cool liquid nourishment should be permitted. If severe pain be present, **bits of ice** may be sucked and **subcutaneous injections of morphin** given. *Chronic catarrh of the esophagus* has been treated by means of **astringent ointments** (argentic nitrate, 0.5—7½ grains; wool-fat and lard, each, 5.0—75 grains), which are applied by means of a sponge-bougie. It is important to direct attention to **causal treatment** (withdrawal of alcohol and tobacco).

PHLEGMONOUS ESOPHAGITIS.

Phlegmonous esophagitis is attended with *suppuration in the submucosa* of the esophagus. The purulent infiltration may also extend to the muscular layer. The pus often ruptures through several openings into the lumen of the esophagus. The disease is difficult of recognition. The most important manifestation consists in sudden disappearance of the signs of *constriction of the esophagus* after *regurgitation of the pus*. Naturally the possibility will always be present that an abscess in the neighborhood of the esophagus—for instance, a burrowing abscess of the vertebral column—has ruptured into the esophagus.

Among the **causes** of this uncommon disorder are impacted foreign bodies, intense corrosion, infectious diseases, extension of inflammatory processes (phlegmonous pharyngitis, phlegmonous gastritis), and suppuration in the neighborhood of the esophagus. At times it is not possible to elicit any cause.

The **treatment** is purely symptomatic.

PEPTIC ULCER OF THE ESOPHAGUS.

Peptic or round ulcer of the esophagus is a rare disorder. The ulcer is situated near the cardia, where it develops in consequence of digestion of the mucous membrane in places by active gastric juice left with food after the act of vomiting. The ulcer is usually round, with sharp margins. It is scarcely recognizable during life. Its dangers consist in hemorrhage, perforation, cicatrization with stenosis of the esophagus, and carcinomatous degen-

SPONTANEOUS RUPTURE OF THE ESOPHAGUS.

A few instances are on record in which rupture of the esophagus has taken place in the midst of perfect health, at times after preceding efforts at vomiting or expulsion, with the occurrence of death within a short time. The patients were usually men addicted to excessive indulgence in alcohol, and probably the wall of the esophagus had been previously softened. As a rule, a longitudinal tear up to five centimeters was present, beginning at the cardia and extending to the lower extremity of the thoracic portion of the esophagus; less commonly two longitudinal tears were present or a single circular tear. As a result the contents of the stomach had entered the mediastinum, and hence the pleural cavity, and the surface of the lung was brownish and softened. The patients, as a rule, have cried out at the onset of the accident that something had ruptured within their body, and have complained of agonizing pain in the course of the vertebral column, with a sense of annihilation and a fear of death, with coldness and pallor of the skin and an imperceptible pulse. Signs of pneumothorax and of subcutaneous emphysema appeared, because air passed from the esophagus into the pleural cavity and into the mediastinal connective tissue, and thence into the subcutaneous connective tissue. There were also nausea, retching, and inability to swallow. Death usually resulted within twenty-four hours, with increasing asthenia.

The **prognosis** is unfavorable.

The **treatment** consists in subcutaneous injections of morphin and camphor in order to relieve the pain and sustain the strength.

SOFTENING OF THE ESOPHAGUS (ESOPHAGOMALACIA).

Softening of the esophagus takes place especially in patients in whom the agonal period is protracted (tuberculous meningitis, diseases of the brain). The wall of the esophagus is swollen and so friable that it tears on the slightest manipulation. Often it is found perforated in the dead subject, and the contents of the stomach have escaped into the abdominal or the pleural cavity. In accordance with the color of the softened tissue, a distinction is made between gray, white, and brown softening. White and gray softening have probably developed only after death, from the entrance of the contents of the stomach into the esophagus, with digestion of its wall. Brown softening may also have occurred during the agonal period, with failure of the circulation, when the acid gastric juice is no longer neutralized by the circulating blood and thereby rendered innocuous. The condition can scarcely be diagnosed during life.

THRUSH OF THE ESOPHAGUS (OESOPHAGOMYCOSIS OIDICA).

Thrush of the esophagus probably occurs always in association with thrush of the oropharyngeal cavity, and is attended with the presence upon the mucous membrane of the esophagus of yellowish or grayish-yellow deposits, in which on microscopic examination the readily recognized thrush-fungus, Oïdium albicans (page 180, Fig. 25), can be found. The deposits, which usually develop abundantly in the upper and lower portions of the esophagus particularly, and do not extend to the mucous membrane of the stomach, are at times punctate, at other times striate, and occasionally they assume the form of hollow or solid casts of the esophagus. As a result they may give rise to difficulty in swallowing. At times, also, plugs of thrush-fungi have been ejected. The mucous membrane of the esophagus is unchanged, or beneath the thrush-deposit there may be observed hyperemia, rarely loss of tissue, purulent infiltration of the submucosa, penetration of the fungi into the blood-vessels, and embolic dissemination to the brain, together with corresponding cerebral manifestations. The affection develops usually in association with thrush of the mouth in debilitated individuals who neglect to keep the buccal cavity clean, and particularly in children with diarrhea and in patients suffering from pulmonary tuberculosis, carcinoma, and diabetes. Should difficulty in swallowing arise, *emetics* may be administered for the purpose of removing the thrush-masses mechanically. Under some conditions *introduction of the sound* will be necessary. In addition, the internal employment of *borax* may be recommended:

℞ Solution of sodium biborate, 5.0 : 200.
Dose: from 5 to 15 c.c. (from 1 teaspoonful to 1 tablespoonful) every two hours.

PARALYSIS OF THE ESOPHAGUS.

Paralysis of the esophagus occurs in association with *disease of the brain and the cervical cord* (hemorrhage, abscess, softening, new-growth, echinococcus, tabes dorsalis, multiple cerebrospinal sclerosis, progressive paralysis of the insane, bulbar paralysis). At times it occurs in conjunction with *hysteria.* It has also been observed in consequence of *pressure on the cervical vagus* by enlarged lymphatic glands. The cases in which the disorder develops in the sequence of diphtheria, syphilis, alcoholism, lead-poisoning, and mercurial poisoning are probably attributable to *neuritic alterations.* At times the affection has appeared after *exposure to cold,* the *ingestion of hot food,* and *psychic disturbances.* The disorder is characterized by *difficulty in swallowing—paralytic dysphagia.* Especial difficulty usually attends the swallowing of fluids and of small particles of food. The act of swallowing is often attended with a peculiar gurgling sound—*dysphagia sonora.* If the patients are careless in the ingestion of food, over-distention of the esophagus therewith may give rise to a *sense of fear, danger of suffocation*, and *palpitation of the heart.* A sound introduced into the esophagus can be moved to and fro with undue readiness and freedom. The *prognosis* depends upon whether the causative conditions can be removed or not. Nourishment should be introduced by means of a *tube*, the paralyzed musculature should be stimulated daily by means of a suitable sound-electrode connected with a *faradic current* of not too great strength and introduced into the esophagus, and the fundamental disorder should be attacked, as, for instance, syphilis with mercury and iodin, diphtheria with injections of strychnin, lead-poisoning with potassium iodid.

SPASM OF THE ESOPHAGUS (ESOPHAGISM).

Spasm of the musculature of the esophagus is observed in some *central neuroses* (chorea, epilepsy, tetanus, hydrophobia, hysteria, hypochondriasis). The disorder may also occur in association with *anatomic diseases of the central nervous system.* At times it is induced by *local irritation of the esophagus* (introduction of the sound, hard articles of food). Those cases in which the condition arises after the employment of belladonna, stramonium, or spoiled meat may be considered as instances of a *toxic* variety. Esophagism occurs through reflex influences in association with enlargement of the tonsils, chronic pharyngeal catarrh, chronic esophagitis, carcinoma of the esophagus and of the stomach, the presence of worms in the intestine, and diseases of the sexual organs. Occasionally it occurs as a result of *psychical disturbances*, as, for instance, in persons who incorrectly believe that they have been bitten by a rabid dog. The principal symptom consists in *diffi-* [illegible] Liquids can be better swallowed than solids. [illegible] deglutition is attended with a disagreeable [illegible], which at times sets in [illegible] The introduction of a [illegible] instrument is suddenly [illegible] award. A dis- [illegible] permitted to remain [illegible] passage of the in- [illegible] obstruction. [illegible] and, in [illegible] persist for hours, [illegible] become pro- [illegible] of the *tube* [illegible] injec- [illegible] of not too great strength may be employed. Of particular importance is the *treatment of the primary disorder.*

IV. DISEASES OF THE STOMACH.

PRELIMINARY CONSIDERATIONS.

Until within a few years it was considered sufficient to apply also to the study of the diseases of the stomach the *physical methods of examination* alone of inspection, palpation, percussion, and auscultation. However valuable the diagnostic results yielded even yet by these methods, they still leave the investigator completely in the dark as to the functions of the stomach, and the fact is therefore to be welcomed that within recent times a *study of the functions of the stomach* has been added to the physical methods of examination. Three conditions especially must be taken into consideration, namely, the absorptive power of the stomach, the motor activity of the stomach, and the constitution of the gastric juice.

1. To determine the **absorptive power of the stomach** a small gelatin-capsule, containing 0.2 (3 grains) of potassium iodid, is employed. This is swallowed by the patient in the morning when the stomach is empty, and the saliva is then examined every five minutes for the presence of iodin. For this purpose starch-paper (bibulous paper that has been dipped in a paste of starch and water and then dried in the air) and fuming nitric acid are used. By touching the mucous membrane of the cheek the paper is moistened with saliva, and then, by means of a glass rod, a drop of fuming nitric acid is applied to the moistened spot. If iodin has been absorbed and has made its appearance in the saliva, the starch-paper is stained reddish or bluish at the exposed spot. In performing this test care should be taken that the gelatin-capsule is wholly swallowed and is not possibly crushed in the mouth, in order that the potassium iodid be not admixed with the saliva already in the mouth. It is absolutely necessary that the test be made when the stomach is empty, because, after food has been taken, the absorptive activity of the stomach is exceedingly variable, even in healthy persons. In a healthy person ten or fifteen minutes will elapse before the first traces of iodin can be demonstrated in the saliva. In the presence of disease of the stomach there is frequently a *prolongation of the period of absorption.* This period is prolonged with especial regularity in cases of carcinoma of the stomach, not rarely for more than sixty minutes. The same condition may, however, be present also in cases of acute and chronic

gastric catarrh, gastric ulcer, and gastric dilatation. The absorptive activity of the stomach is impaired, further, in anemic, cachectic, and febrile states.

Little is known with regard to *acceleration of gastric absorption*. It is believed to have been observed in cases of recent ulcer of the stomach, without cicatrization.

2. The **motor activity of the stomach** is studied by having the patient take a *test-meal*, and determining after an interval of six hours, with the aid of a soft *tube* introduced into the stomach, whether remains of the food are still present or not. If the motor activity of the stomach is unimpaired, the viscus will have propelled its contents into the intestine within six hours. A *test-meal* consists of a plate of soup made with water or with flour, a tender beefsteak with an egg, and a wheat roll. An ordinary rubber gas-tube may be employed as a *stomach-tube*, the sharp edges being cut away with the aid of scissors, or a soft stomach-tube with lateral fenestra, which is virtually nothing more than an unusually long and thick Nélaton catheter. Both varieties of tube can be readily introduced into the stomach with swallowing movements. In order to obtain the gastric contents the *expression-method* may be employed. After the tube has been introduced into the stomach down to the lower curvature the patient begins to make active expulsive efforts. If the stomach contains chyme, this will be expelled, and it can be received into a vessel held beneath the free opening of the tube. Should unexpectedly nothing appear, a glass funnel must be introduced into the free opening of the stomach-tube, and through this lukewarm water should be introduced into the stomach. As soon as the flow ceases the introduction is suspended, the tube is pinched between the fingers, the glass funnel is inverted into a suitable vessel, and the pressure of the fingers is released, when the water and the gastric contents will escape. *Diminution in the motor activity of the stomach* may be recognized from the fact that the viscus still contains considerable amounts six hours after the ingestion of food. The condition occurs especially in association with carcinoma, dilatation, and atony of the stomach, but it may also attend other diseases of the stomach, as, for instance, gastric catarrh.

Little is known with regard to *morbid increase in the motor activity of the stomach*, although a number of observations are on record in which the stomach was empty two hours after the ingestion of food, and the patient constantly complained of hunger. It should further be noted whether the gastric contents obtained exhibit evidence of insufficient mastication of the food—a common fault and a frequent cause of diseases of the stomach of varied kind.

3. The **digestive power of the gastric juice** depends upon the [illegible] hydrochloric acid, pepsin, and lab-ferment are [illegible] to all of these in cases

of disease of the stomach. In order to obtain gastric juice for examination a *test-meal* or a *test-breakfast* is given. A test-meal may be constituted of the ingredients previously named, viz., a plate of soup made with water or with flour, a tender beefsteak with an egg, and a wheat roll. The test-breakfast consists of a large cup (250 c.c.) of tea without sugar and of a wheat roll (35 gm.). The gastric contents are obtained by expression with the aid of a soft stomach-tube four hours after, and a test-meal one hour after, a test-breakfast. The periods named have been shown by experience to be the most favorable for the examination. Personally, I prefer the test-breakfast to the test-meal, because the manipulations are cleaner and the examination can be more quickly terminated without affecting its reliability. The contents of the stomach obtained are naturally not pure gastric juice, but chyme mixed therewith; experience has, however, shown that under normal conditions this somewhat dilute gastric juice possesses quite definite and constant properties.

In the first place, the materials obtained are carefully inspected with the unaided eye for the presence of foreign admixture (blood, mucus, shreds of tissue), and with regard to the condition of the food ingested. Then the reaction of the gastric juice is tested by means of *litmus-paper*. This will be found almost unexceptionally acid, so that blue litmus-paper is stained red. The presence of an acid reaction by no means indicates that the gastric contents contain free acid, for it may be due also to acid salts in solution, especially phosphates. Next, Congo paper (bibulous paper stained with a solution of Congo red) is used to determine whether the gastric contents contain free acid. In this event the Congo paper is stained blue. The Congo reaction yields no information as to the nature of the free acid, and whether it be due to hydrochloric acid or to organic acids (acetic, lactic, butyric).

To demonstrate the presence of free hydrochloric acid the *phloroglucin-vanillin solution* of Günzburg (phloroglucin, 2.0; vanillin, 1.0; alcohol, 30.0) is employed. Of this reagent about 5 drops are placed upon a porcelain dish, and an equal amount of filtered gastric contents is added. The mixture is then carefully heated over a flame until dried. If the gastric juice contain free hydrochloric acid, a beautiful carmine-red color appears on evaporation. Complete investigation of the gastric juice requires further also *quantitative determination of the hydrochloric acid.* It is necessary to understand that two kinds of hydrochloric acid must be distinguished in the gastric contents, the *combined* and the *free hydrochloric acid.* The proteids of the gastric contents immediately enter into combination with a portion of the hydrochloric acid of the gastric juice, and only when this combination has been completely saturated does hydrochloric acid remain uncombined, therefore, as free hydrochloric acid. In the further digestion of proteids this free hydrochloric acid only is of importance, and this fact will explain why attention has been directed especially, and often even exclusively, to the free hydrochloric acid. Experience has shown that in a healthy person after ingestion of the test-meal previously mentioned the proportion of free hydrochloric acid in the gastric juice is from 0.15 to 0.25 per cent.

The chemic methods for determining the total hydrochloric acid, the combined and the free hydrochloric acid in the gastric juice, are too complicated for the general practitioner, apart from the fact that they cannot even be considered as accurate, and often it will suffice for the physician to estimate the *total acidity of the gastric juice*, which can be done without much trouble or the consumption of much time. In doing this not only the hydrochloric acid must be considered, but everything in solution in the gastric contents that yields an acid reaction, as, for instance, phosphates, and under some conditions lactic and acetic and butyric acids. To determine the *total acidity of the gastric contents* a 1 : 10 normal sodium-hydroxid solution is employed, of which 1 c.c. neutralizes 0.00365 gm. of hydrochloric acid. The test is made in the following manner: 10 c.c. of filtered gastric contents are introduced into a glass by means of a pipet, and 5 drops of an alcoholic solution of phenolphthalein are added. The gastric contents remain unchanged; at most a slight and entirely insignificant cloudiness forms. From a graduated buret normal sodium-hydroxid solution is permitted to drop until a coloration appears, and persists in spite of constant shaking. By this means evidence is furnished that the first drop of normal sodium-hydroxid solution is in excess, and that all of the acid of the gastric contents is neutralized, for a phenolphthalein solution that remains colorless in an acid solution at once assumes a red hue with the slightest excess of alkali. The estimation is made as follows:

If 4 c.c. of normal sodium-hydroxid solution are employed,
1 " sodium hydroxid = 0.00365 of hydrochloric acid,
4 " " " = 0.01460 " " and
10 " gastric contents contain 0.0146 " " and
100 " " " " 0.146 " " = 0.146 per cent.

If, continuing the illustration, 4 c.c. of sodium-hydroxid solution have been required to neutralize 10 c.c. of gastric contents, and therefore 40 c.c. of sodium hydroxid to neutralize 100 c.c. of gastric contents, the total acidity of the gastric juice is for brevity's sake designated simply as 40. The gastric contents of a healthy individual should have a total acidity of from 30 to 60. If the total acidity be less than 30, the condition is designated hypacidity, and if more than 60, hyperacidity.

Hypacidity is inconceivable without deficiency of free hydrochloric acid in the gastric juice, so that the demonstration of total hypacidity indicates also hypochlorhydria. Hyperacidity of the gastric juice can, however, be induced also by increase in the organic acids. Under such circumstances the free hydrochloric acid and the organic acids must be determined quantitatively. For this purpose the filtered gastric contents are shaken vigorously with an equal amount of ether in a separatory funnel, and the gastric contents are then poured back into a glass vessel. The ether will take up the organic acids. Then normal sodium-hydroxid solution is added to 10 c.c. of gastric contents until the first drop of gastric contents heated upon a porcelain dish with a drop of phloroglucin-vanillin solution no longer yields a red color. In this way demonstration would be afforded that all free hydrochloric acid has been neutralized by the normal sodium-hydroxid solution. The amount of hydrochloric acid can be readily estimated from the amount of sodium-hydroxid solution employed.

If it be further desired to estimate the amount of organic acids, it would be necessary to evaporate the ether, to dissolve the residue in water, and to determine the acidity of the aqueous solution by means of normal sodium hydroxid and phenolphthalein in the manner previously described. Organic acids are generated in the stomach by the fermentation of carbohydrates. *Lactic acid* is almost constantly present in the gastric contents. It can be readily demonstrated with the aid of *Uffelmann's reagent* (solution of ferric chlorid, 1 drop; carbolic acid, 0.4; distilled water, 30.0). If filtered gastric juice be added to Uffelmann's reagent, a canary-yellow color appears in the amethyst-blue solution in the presence of lactic acid. Hydrochloric, acetic, and butyric acids likewise induce discoloration, but the color is smoky gray and not yellow. For the recognition of *acetic* and *butyric acids* dependence will generally be placed upon the sense of smell. A penetrating sour odor is indicative of the presence of acetic acid, and a rancid odor of that of butyric acid.

The determination of the hydrochloric acid in the gastric juice is naturally of great significance in treatment. Originally it was thought that the knowledge was of great diagnostic importance, but this has not been confirmed by more extensive experience. Anachlorhydria occurs frequently, but not constantly, in association with carcinoma of the stomach. It is also observed in connection with acute and chronic gastric catarrh, extensive cicatrization of the gastric mucous membrane after the action of corrosive agents, amyloid degeneration of the gastric mucous membrane, and anemic, cachectic, and febrile states. Hyperchlorhydria has been observed in connection with round ulcer of the stomach, and it also occurs as an independent gastric neurosis.

Among *organic acids* the abundant and persistent appearance of lactic acid has erroneously been given diagnostic significance with regard to the presence of carcinoma of the stomach. Organic acids may be abundantly present in connection with all fermentative processes of the gastric contents, and with especial constancy with gastric dilatation.

Pepsin occurs almost always in sufficient amount in the gastric juice. It is only wanting, together with hydrochloric acid, when the glands of the gastric mucous membrane have undergone atrophy—so-called gastric anadenia. In order to form an opinion as to the sufficiency of the pepsin in the gastric contents the so-called *four-glass test* is made. Into each of four test-tubes are introduced 5 c.c. of filtered gastric contents. The first tube is left unchanged, to the second are added 0.5 of pepsin, to the third 2 drops of dilute hydrochloric acid, and to the fourth 0.5 of pepsin and 2 drops of dilute hydrochloric acid. Then a disc of albumin of uniform size, best obtained by means of a cork-cutter, or flakes of fibrin of equal amount, are introduced into each tube. The fibrin should be thoroughly washed and freed from blood, and it can then be kept in glycerin. The four tubes are placed in the thermostat at the temperature of the body, and the digestive process during the succeeding hours is observed. If satisfactory digestion takes place in the first tube, the result indicates that the gastric juice contains sufficient free hydrochloric acid and pepsin. If digestion takes place in the second test-tube and not in the first, this indicates a deficiency of pepsin; if in the third test-tube and not in the first or second, a deficiency of hydrochloric acid; and if only in the fourth tube, a deficiency of both pepsin and hydrochloric acid.

The gastric juice almost always contains *lab-ferment* also in sufficient amount. This is absent only in gastric adenia. To demonstrate its presence, to 10 c.c. of unboiled milk 5 drops of filtered gastric contents are added, and the mixture is placed in a thermostat at the temperature of the body. The presence of lab-ferment is indicated by the occurrence of coagulation in the milk within from fifteen to thirty minutes.

ACUTE GASTRIC CATARRH.

Etiology.—Acute gastric catarrh is an exceedingly common disorder, and in the majority of cases it is dependent upon **errors in diet.** These may at times consist in the ingestion of too full a meal, at other times in the ingestion of food that is too hot or too cold or insufficiently masticated, and therefore generally eaten too hastily, and at still other times finally in the ingestion of spoiled food or drink. Infants are frequently attacked by acute gastric catarrh, because milk is readily decomposed and frequently contains fermentative bacteria. All other causative factors are much less commonly effective. Gastric catarrh is, however, most likely to be of **toxic** origin, less commonly from the ingestion of chemic poisons than from indulgence in alcohol in too great concentration or in too great amount. *Traumatic gastric catarrh*, resulting from injury in the epigastrium, and *rheumatic* or *refrigeratory gastric catarrh*, resulting from the local or general influence of cold, are of rare occurrence. **Acute infectious** and **debilitating diseases** generally are more frequently accompanied by acute gastric catarrh. Gastric catarrh may arise by extension from adjacent disease, as, for instance, in connection with intestinal catarrh or peritonitis.

Anatomic Alterations.—Little is definitely known with regard to the anatomic alterations of acute gastric catarrh in human beings, because opportunity is seldom afforded for post-mortem examination. The principal alterations usually involve the pyloric region, and consist in redness and swelling of the mucous membrane and increased secretion of mucus. Microscopically granular cloudiness of the main cells, the glands, hyperemia of the blood-vessels, and proliferation of round cells in the gastric mucous membrane will be apparent.

Symptoms and Diagnosis.—Among the symptoms of acute gastric catarrh the most constant is **loss of appetite—anorexia.** In some cases there is a desire for piquant and highly salted or acid articles of food particularly. **Thirst** is usually increased. Not only the sight, but also the thought, of food excites disgust and nausea. Often **vomiting** occurs, the food not rarely being expelled in a state of fermentation and decomposition. When vomiting is repeated the ejected matters not rarely acquire a greenish color from admixture with biliary coloring-matter. Often the patients complain that the vomited matters possess a bitter taste, which may be due not alone to the biliary coloring-matter, but also to the peptone present. In less severe cases, or in connection with previous vomiting, there occurs frequently **eructation—singultus.**[1] Often acid or rancid gases are thus expelled, which at times diffuse an odor of hydrogen sulphid. Acid gastric contents also are sometimes brought up, leaving in the epigastrium or in the lower segment of the esophagus a burning, boring sensation, which is known as **heartburn—pyrosis.**

Local examination of the stomach usually discloses extensive **tenderness on pressure in the epigastrium,** which is at times distended with gas like an air-cushion. The patients also often complain of a sense of tension, of pressure, or of pain in the epigastric region. The **functions of the stomach** are altered in so far as the secretion of hydrochloric acid ceases, while mucus is produced in increased amount. The motor activity of the stomach is diminished, and the food therefore remains in the stomach for an unduly long time. The **tongue** is usually coated, and a **disagreeable odor** often emanates **from the mouth.** The patient frequently complains of a **pasty taste.** Frequently the **bowels are irregular,** sometimes constipation, sometimes diarrhea, existing. The **urine** is not rarely voided in small amount, presenting a deep-red, highly concentrated or saturated color, and frequently depositing a brick-dust sediment of urates. The disorder is at times attended with slight **fever—gastric fever.** It is noteworthy that the **general condition** may be greatly disturbed. Vertigo, mental confusion, and headache are frequently recurring symptoms, and are perhaps to be attributed to the action upon the central nervous system of toxins formed in the stomach. The frequent occurrence of *facial herpes*, especially labial herpes, is perhaps also due to toxic effects upon peripheral nerves. The *duration of acute gastric catarrh* varies between days and one or two weeks. Relapses are extremely common in the presence of recurring injurious influences, and these readily lead finally to the development of chronic gastric catarrh.

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[1] In English, eructation is synonymous with belching, and singultus with hiccup.—A. A. E.

Prognosis.—Acute gastric catarrh is not a fatal disease, and the prognosis is therefore favorable. With the coöperation of the patient relapses can be generally avoided.

Treatment.—In the treatment of diseases of the stomach in general and particularly in that of acute gastric catarrh the greatest importance should be attached not to the administration of numerous medicaments, but to appropriate **dietetic regulations.** Solid food should be entirely prohibited, and only weak tea, broths, milk-soup, and mucilaginous soups permitted. Abstinence from food is the best treatment. **Causal indications** are present when the stomach has been overloaded. The viscus should then be emptied by means of an emetic, most serviceably by apomorphin hydrochlorate (0.2 : 10 ; 8 minims subcutaneously). If the sense of pressure and of pain be excessive, a **hot cataplasm** should be applied to the epigastrium. **Symptomatically,** *dilute hydrochloric acid* (10 drops in a wine-glassful of tepid water half an hour after meals) may be prescribed. For the relief of *heartburn alkalies*, as, for instance, sodium bicarbonate (from 10 to 20 grains) or lime-water (1 tablespoonful) or mineral waters, may be given. Vertigo and headache may be relieved by *antipyrin* or *phenacetin* (1.0—15 grains—thrice daily).

CHRONIC GASTRIC CATARRH.

Etiology.—**Errors in diet** are also the most common cause of chronic gastric catarrh. Irregularity and undue haste in eating are especially important, so that the food is not sufficiently masticated. Those engaged in certain pursuits (merchants, lawyers, physicians) suffer from this disorder with especial frequency, because the influences named are particularly operative among them. **Defective teeth** are also responsible for the entrance of the food into the stomach in an imperfectly prepared state. Not rarely acute gastric catarrh if frequently repeated gradually passes into the chronic form. An unfortunately common variety of chronic gastric catarrh is that known as *drunkards' catarrh*, from which those who indulge in alcohol to excess frequently suffer. *Heavy* *smokers* also are not rarely the victims of chronic gastric catarrh. Under both of the last-named conditions **toxic influences** are operative. *Hypostatic gastric catarrh* occurs especially in association with chronic diseases of the respiratory and circulatory organs, and with diseases of the liver, especially cirrhosis. *Cachectic* and *anemic gastric catarrh* develop in the course of chronic disease attended with impoverishment of the blood and wasting discharges. At times chronic gastric catarrh occurs in conjunction with *other diseases of the stomach*, as, for instance, carcinoma. From the nature of the causative factors chronic gastric catarrh is *most common in men*, and it occurs almost exclusively in adults.

Anatomic Alterations.—The anatomic alterations of chronic gastric catarrh involve the **pyloric region** by preference. The mucous membrane is usually covered with an abundance of mucus, which in places is turbid and pus-like, and may even be bloody or brownish in color. In addition the mucous membrane presents a brownish-red appearance, and it exhibits here and there dilated vessels and extravasations of blood. At the same time the mucous membrane is notably swollen and thickened.

On *microscopic examination* the superficial epithelium of the gastric mucous membrane will be found involved in marked mucoid degeneration. The glandular cells exhibit granular turbidity, are in part shrunken and uniform, so that the differentiation between the parietal cells and the chief cells is lost. Accumulation of round cells in greater or lesser number has taken place between the gland-tubules. The blood-vessels are dilated and greatly distended. Not rarely remains of blood-pigment are present, indicating preceding hemorrhage.

Chronic gastric catarrh readily gives rise to inflammatory hyperplasia of the submucosa, and even of the muscularis and the serosa, and under such conditions the designation *hypertrophic gastric catarrh* may be employed. In consequence of excessive hyperplasia of the submucosa in places the mucous membrane sometimes presents numerous wart-like elevations, and there results the condition described as *état mamelonné*. At times the hyperplastic tissue undergoes cicatricial contraction, and the stomach undergoes the change known as *cirrhosis*. If circumscribed hyperplasia of the mucous membrane take place, there form under some conditions numerous pendulous tumors, or gastric polypi—so-called *chronic polypous gastritis*. Excessive proliferation of round cells between the glands may cause atrophy of the latter by pressure, and, if at the same time, in consequence of preceding hemorrhage, much gray or blackish pigment be present in the mucous membrane, the condition is designated *atrophic pigmentary induration of the gastric mucous membrane*. At times almost the whole of the glandular structure is destroyed, so that the mucous membrane resembles granulation-tissue, and the condition is then known as *chronic atrophic gastritis*, or *phthisis* *ventriculi*, or *gastric anadenia.* Finally, it may be that some glands, after occlusion of the excretory duct, are transformed into cystic cavities—so-called *chronic cystic gastritis.*

Symptoms and Diagnosis.—The symptoms of chronic gastric catarrh resemble in many respects those of acute gastritis, but the peculiarities of an acute disorder are wanting. **Alterations in appetite** are among the most constant symptoms. Usually there is anorexia, but at times there may be bulimia, and especially a desire for piquant articles of food. **Thirst** is often, but not always, increased. Generally the **tongue** presents a grayish or brownish deposit, and the patient often complains of an unpleasant and pasty **taste.** Not rarely a **disagreeable odor emanates from the mouth.** At times there is **increased secretion of saliva.** Frequently there is troublesome **eructation—singultus,** which often is followed by **heartburn—pyrosis.** The gases brought up by eructation not rarely taste and smell rancid or acid. **Vomiting** occurs less commonly with chronic than with acute gastric catarrh. The vomited matters often consist of rancid or acid fermenting gastric contents, in which microscopic examination discloses the presence of yeast-fungi and sarcinæ. Drunkards frequently suffer in the morning, when the stomach is empty, from distressing retching and vomiting of masses resembling saliva, and which consist of saliva swallowed during the night, and less commonly of carbohydrates that have undergone mucoid fermentation. This manifestation is known as the *morning vomiting of drunkards.* The **epigastrium** is not rarely tender upon pressure throughout an extensive area. The **functions of the stomach** are variously altered. The motor function of the stomach is impaired with especial constancy, so that the food is retained in the stomach for an unduly long time and readily undergoes decomposition. Absorption on the part of the gastric mucous membrane also is almost always delayed. In the less severe cases (simple chronic gastric catarrh) the secretion of hydrochloric acid is diminished, while in the more severe cases (chronic mucous gastric catarrh) free hydrochloric acid is wanting in the gastric contents. When anadenia exists, not only is hydrochloric acid wanting, but also pepsin and lab-ferment. The **bowels** are usually constipated and irregular. **Urine** is generally voided in small amounts; often on cooling a red granular, brick-dust sediment of urates is precipitated.

Frequently **mental manifestations** are associated with chronic gastric catarrh. The patients become hypochondriacal, prefer to be alone, are disinclined to mental and physical activity, and oppress themselves with the most dismal reflections as to their future. Depression, apprehensions and fear of places—agoraphobia—also occur. Other complaints are made of mental confusion, a sense of [illegible] or headache. Vertigo occurs with especial frequency [illegible] which usually is particularly noticeable in the morning. Some patients complain of *palpitation of the heart* and of irregularity in the action of the heart. Asthmatic conditions arise also in connection with marked distention of the stomach with gas—*dyspeptic asthma.*

Chronic gastric catarrh is unattended with fever. At times *emaciation and earthy pallor* become so pronounced that latent gastric carcinoma is rather suspected. Anemia of high grade with its sequels develops in conjunction with gastric anadenia, and the clinical picture may then entirely resemble that of progressive pernicious anemia.

The *duration of the disease* is extremely variable. Some patients suffer for years, and even throughout life, from chronic gastric catarrh. Among the *sequels dilatation of the stomach—gastrectasis*—may be mentioned particularly, but *carcinoma of the stomach* also is by no means rarely a sequel of chronic gastric catarrh.

Prognosis.—The prognosis depends not rarely upon the coöperation of the patient, and upon his willingness to forego excesses in eating and in the use of alcohol and tobacco. Incurable causative factors naturally render the prognosis unfavorable.

Treatment.—The treatment should be in the first place **causal** and at the same time **prophylactic.** The patient should be advised to eat slowly and with regularity, taking care to masticate the food thoroughly and to keep the teeth in good condition. Drunkards must give up the use of alcohol, and smokers be restricted in that of tobacco. The **symptomatic treatment** depends upon the nature of the gastric disturbances. If the gastric contents are free from hydrochloric acid, *dilute hydrochloric acid* (10 drops in a wine-glassful of tepid water half an hour after meals) should be prescribed. To the hydrochloric acid from 5 to 10 grains of *pepsin* should be added, if this also be absent from the gastric juice. If active fermentation take place in the gastric contents, systematic *irrigation of the stomach* by means of a siphon-arrangement should be practised. Only rarely will it be possible to correct gastric fermentation by the internal administration of *antizymotics*, as, for instance:

℞ Salicylic acid, 0.5 (7½ grains);
Saccharin, 0.02 (⅓ grain).—M.
Make 10 such starch-capsules.
Dose: 1 capsule thrice daily half an hour before eating.

Or,

℞ Resorcin, 0.1 (1½ grains);
Saccharin, 0.02 (⅓ grain).—M.
Make 10 such powders.
Dose: 1 powder thrice daily half an hour before eating.

Prolonged stasis of food in the stomach may at times be overcome by administration of *bitters*, which improve the tone of the gastric musculature, as, for instance:

℞ Strychnin nitrate, 0.1 (1½ grains);
Powdered althea-root, sufficient to make 15 pills.
Dose: 1 pill thrice daily.

Or,

℞ Wine of condurango, 200.0.
Dose: 1 tablespoonful thrice daily.

Water-cures are much employed, and those that can be carried out at bathing-resorts, where the diet is strictly regulated, are especially commendable. Carlsbad serves as an example. In general, alkaline mineral springs (Neuenahr, Vichy, Bilin, Giesshübel, Fachingen), alkaline-chlorin springs (Ems, Selters, Gleichenberg), alkaline-saline springs (Carlsbad, Marienbad, Franzensbad, Tarasp), and sodium-chlorid springs (Wiesbaden, Homburg, Kissingen) are worthy of consideration. Residence in the mountains and pedestrian tours in the mountains also may be recommended, and they frequently exert a useful influence upon the mental state.

SUPPURATIVE INFLAMMATION OF THE STOMACH (PURULENT OR PHLEGMONOUS GASTRITIS).

Purulent gastritis is an extremely rare disease. It is attended with *suppuration in the submucosa*, the pus either infiltrating the tissues in a diffuse manner or collecting in a circumscribed manner into an abscess. At times the pus has been partially evacuated into the cavity of the stomach or into the peritoneal cavity, and perforation may take place through numerous cribriform openings. Often purulent peritonitis develops. Signs of general septicemia, especially enlargement and softening of the spleen, are frequently observed. The causes are either of *infectious* or of *toxic nature*. Thus, phlegmonous gastritis is at times observed to develop in the sequence of *infectious diseases*. At other times it is associated with other *diseases of the stomach*—as, for instance, carcinoma—or the inflammatory process has extended from the pharynx or the esophagus to the submucosa of the stomach. *Poisoning* with corrosive acids and alkalies, and excessive indulgence in *alcohol* may also be mentioned as causes. Recognition of the disease is scarcely possible. It is often concealed behind the clinical picture of *general septicemia* or *peritonitis*. Fever, epigastric pain, and vomiting are particularly constant manifestations. The disorder would be most readily recognized if a tumor in the epigastrium becomes smaller or disappears after the vomiting of pus. Death usually occurs within a few days amid signs of progressive asthenia. Recovery with cicatrization probably takes place but rarely.

The **treatment** consists in the administration of stimulants, the swallowing of bits of ice, and the application of an ice-bag to

ROUND ULCER OF THE STOMACH.

Etiology.—Round ulcer of the stomach is a *common disorder* that in some regions attacks more than 5 per cent. of the population. Childhood is usually exempt from the disease, while the age between the fifteenth and the thirtieth year of life suffers especially. *Women* are attacked more than twice as commonly as men. The disorder develops with striking frequency in the sequence of **chlorosis,** in consequence, it is thought, of anemic fatty degeneration of the blood-vessels of the stomach, and thrombosis in the diseased areas. At times the **ingestion of hot articles of food** is followed by the formation of a round ulcer of the stomach. The disease is, therefore, observed with unusual frequency in cooks. Traumatism in the epigastric region, as, for instance, a severe blow, may act as a cause of the disorder. In this group of etiologic factors may be included also violent **efforts at vomiting,** which may give rise to hemorrhage and to hemorrhagic erosion of the gastric mucous membrane, and secondarily to gastric ulceration. Certain **infectious diseases,** particularly pulmonary tuberculosis and syphilis, have been made responsible for the development of round ulcer of the stomach, but this supposition still requires confirmatory evidence. At times round ulcer of the stomach, like ulceration of the duodenal mucous membrane, has been observed to develop after extensive **burns of the skin,** perhaps in consequence of thrombosis of gastric vessels by blood-plates. Further, the predisposition to round ulcer of the stomach may be augmented by certain injurious influences acting upon the stomach, among which may be named the ingestion of starchy food and of alcohol in excessive amounts.

Anatomic Alterations.—The round ulcer of the stomach acquires its name from its shape. It is generally round, and if it is confined to the gastric mucous membrane it presents an appearance as if the mucous membrane in the diseased situation had been cut out with a punch. It is, therefore, characterized by its sharp borders, which usually are but little elevated, and on microscopic examination exhibit slight alterations (round-cell infiltration). The muscular layer of the stomach is not rarely exposed in the floor of the ulcer, as if it had been artificially dissected out with a knife. The ulcer *varies in size.* At times it is so small that it may be readily overlooked, while at other times it may attain the size of the palm of the hand. It is most frequently *situated* in the neighborhood of the pylorus and upon the posterior wall of the stomach, and least commonly at the cardia. Usually but a single ulcer is present. If there are several, they may be in various stages of development. At times contiguous ulcers coalesce, and as a result irregular excavated losses of tissue take place. Not rarely the ulcerative process extends from the mucous membrane to the muscular layer, and even to the serosa. Such a condition is attended with *danger of rupture of the stomach*, and this accident occurs with such frequency that the round ulcer of the stomach has also been designated perforating ulcer of the stomach. Fortunately, free rupture of the stomach into the peritoneal cavity is frequently averted by the formation of adhesions between the stomach and adjacent organs, as with the liver, the pancreas, or the spleen, as a result of previous circumscribed peritonitis. At the same time the destructive process may also extend to these organs, and give rise to alarming hemorrhage by perforation of large blood-vessels. The borders of deep ulcers are sometimes terrace-like, and usually steeper upon one side than upon the other. The ulcer is sometimes funnel-shaped, with its broad base directed toward the gastric mucous membrane. The apex of the funnel, however, is not situated vertically over the center of the base, but eccentrically. The entire shape of the ulcer is suggestive of the mode of division of the gastric vessels, and the impression is created as if the distribution of a gastric artery had been involved in the ulcerative process. If a gastric ulcer undergoes *cicatrization*, the mucous membrane in the vicinity of the margin of the ulcer is often thrown into radiating folds. Gastric cicatrices at the pylorus not rarely are followed by stenosis at this orifice and dilatation of the stomach. By contraction cicatrices may give rise to deformities of the stomach. At times cicatrices at the middle of the stomach may cause the stomach to assume the shape of an hour-glass.

Symptoms and Diagnosis.—Occasionally gastric ulcer is wholly unattended with symptoms, and is accidentally discovered on post-mortem examination—*latent gastric ulcer*. In other instances the development of a gastric ulcer is insidious, but hemorrhage or perforation takes place suddenly, and may cause death within a short time. Among all the symptoms of round ulcer of the stomach, **hemorrhage from the stomach—gastrorrhagia**—is the most reliable in diagnosis. In its absence the diagnosis will only be more or less probable. Hemorrhage from the stomach takes place, however, in but one-third of the cases, and its occurrence depends upon whether vessels of considerable size are perforated by the ulcerative process before thrombosis has taken place. Most frequently hemorrhage from the stomach gives rise to the **vomiting of blood—hematemesis.** The vomited blood appears as dark-red, partly coagulated masses, is often admixed with particles of food, and usually yields an acid reaction. The loss of blood may be so considerable as to render death imminent. The patients present a cadaveric pallor, with a soft, small, and dicrotic pulse, feeble heart-sounds, often systolic heart-murmurs, and dilatation of the right ventricle. Not rarely albuminuria develops, with slight edema about the ankles, and even edema of the face. In consequence of cerebral anemia attacks of syncope occur, especially upon assuming the upright posture, with roaring in the ears, impairment of hearing, blurring of vision, palpitation of the heart, and shortness of breath. Death takes place amid chronic convulsions. At times blindness follows recovery from gastric hemorrhage.

Much less commonly hemorrhage from the stomach is attended with coffee-ground, ink-like, or soot-like vomiting, which, on the other hand, is common with gastric carcinoma. It occurs with gastric ulcer when but small hemorrhages have taken place, and the blood remains in the stomach for a considerable time, in consequence of which the hemoglobin is converted into hematin by the hydrochloric acid of the gastric juice.

Far less commonly hemorrhage from the stomach is unattended with hematemesis, but discharge of blood takes place from the bowel, as manifested by the appearance of **black, tarry stools.** This occurrence must always be thought of when patients suddenly lose consciousness at stool and become pale. Not rarely slight febrile movement occurs, probably in consequence of absorption of decomposed blood from the intestine into the circulation. It is rare for hemorrhage from the stomach always to be manifested by bloody stools alone. More frequently bloody vomiting occurs in conjunction with bloody stools.

Epigastric pain—gastralgia—is an exceedingly common symptom of gastric ulcer, but it is of varied significance. Pain in the epigastrium appears usually a short time after the ingestion of food, and is readily induced by inappropriate food. Palpation of the stomach also induces pain, and especial consideration should be given to the fact that this pain is confined to a circumscribed area. At times pain in the stomach appears in certain postures, as, for instance, the lateral decubitus. The **functions of the stomach** exhibit no peculiarity. The period of absorption is generally prolonged, rarely shortened. The hydrochloric acid of the gastric juice is often, but by no means constantly, increased (more than 0.25 per cent.). The motor functions of the stomach exhibit no alteration. The patients often complain of frequent and persistent **vomiting.** Some scarcely retain food for weeks and months, and alarming emaciation may result. **Eructation** and **pyrosis** are not uncommon symptoms. The **appetite** is usually wanting, while **thirst** is increased. In patients in whom vomiting is frequent the **tongue** often presents a vivid-red color and a clean appearance, probably in consequence of irritation due to the marked acidity of the vomited matters. Individuals with round ulcer of the stomach, as a rule, emaciate rapidly and acquire a pallid appearance. Often they become extremely nervous and sleepless. Even when cicatrization of the ulcer takes place, they must frequently be careful in eating and drinking throughout life.

The *duration of the disease* usually extends over several months, whence the name chronic gastric ulcer. Besides, there is a great *tendency to recurrence*, so that some patients, in spite of all care, are annoyed and alarmed from time to time by renewed symptoms of gastric ulceration. Among the *complications* of round ulcer of the stomach the most frequent, and diagnostically the most important, has already been mentioned, namely, *hemorrhage from the stomach*. Another serious complication consists in *peritonitis* and *perforative peritonitis*. Simple peritonitis is generally confined to the immediate vicinity of the ulcer. Perforative peritonitis as a rule sets in abruptly, and gives rise to alarming collapse. The abdomen becomes distended, tense, and universally tender on pressure, hepatic and splenic dulness have disappeared, and death results often within a short time.

Perforation of the ulcer takes place less commonly into the bowel, through the diaphragm into the pleural or the pericardial cavity, and even into the lungs or into the heart, than into the abdominal cavity. A subphrenic abscess may also develop in consequence of perforation of a gastric ulcer.

Among *complications gastralgia* and *great sensitiveness of the stomach* should be especially mentioned, and these may occasionally persist throughout life. Cicatrized ulcers at the pylorus may give rise to *pyloric stenosis* and *dilatation of the stomach*. Destruction of the muscular ring of the pylorus is followed by *pyloric insufficiency*. Especial significance should be attached to the fact that at times in advanced life *carcinoma* develops at the border of a gastric ulcer.

Opinions are divided as to the *pathogenesis of round ulcer of the stomach*. In our opinion local disturbances in the circulation in the gastric mucous membrane constitute the first alteration. In favor of this view is the shape of the ulcer, which often reproduces the area of distribution of a blood-vessel of moderate size. At times thrombotic occlusion may have taken place, at other times rupture of a blood-vessel with hemorrhage. The portion of gastric mucous membrane cut off from its blood-supply may be digested by the gastric juice, a process that is prevented under normal circulatory conditions by the alkalinity of the blood. If in addition hyperacidity of the gastric juice be present, digestion is favored in a still greater degree. The round ulcer of the stomach may, therefore, be designated a peptic ulcer. Possibly bacteria also take some part in the destruction of the gastric mucous membrane.

Prognosis.—The prognosis of round ulcer of the stomach is always grave, for of the most dangerous complications there is no small number, and these may occur unexpectedly in spite of all watchfulness over the patient.

Treatment.—In the treatment of round ulcer of the stomach diet plays an important part. As long as the ulcer has not undergone cicatrization the patient should remain in bed and keep a hot cataplasm constantly applied to the epigastrium. Only liquid and unirritating food should be permitted. A milk-diet is the best. The milk, after thorough boiling, should be taken in small swallows every quarter of an hour to the amount of one or one and a half liters per day. For patients who have an aversion to milk, weak tea, coffee, or meat-broth may be added. Should milk not be well borne, it can be made into a thin soup with the aid of fine flour; and should it undergo fermentation and acid decomposition readily in the stomach, a tablespoonful of lime-water may be added to each glass. Some persons bear buttermilk better, and it will then be advisable to prescribe a course of **buttermilk-treatment.** If the stomach is exceedingly sensitive to the ingestion of food, it may be necessary for a time to forbid all food by the mouth, and to confine the feeding to **nutrient enemata.** The simplest mixture for this purpose consists of equal parts of milk and slightly salted meat-broth, to which some sugar, 2 raw eggs, and 10 drops of tincture of opium may be added. This mass, at the temperature of the body, is permitted to flow slowly into the rectum thrice daily in amounts up to 300 c.c. by means of a Hegar funnel, after the rectum has previously been cleared of fecal matter by irrigation. The resumption of solid food should be effected slowly. There may then be administered soft-boiled eggs, meat-peptone, calves' brain, scraped beef, scraped ham, scraped sirloin, veal-cutlets, rice-pap, potato-pap. With regard to vegetables, fruits, and fatty articles of food, care must be taken for a long time, often throughout the remainder of life. Among medicaments we would especially recommend **artificial Carlsbad salt** and **bismuth salicylate.** Of the salt, 1 or 2 teaspoonfuls should be dissolved in 500 c.c. of tepid water, and this should be drunk slowly every morning for four weeks while the stomach is empty. The object aimed at is to diminish the hyperacidity of the gastric juice, to remove from the gastric mucous membrane such mucus as may be present, and to induce evacuation of the bowels. The bismuth salicylate is intended to favor the process of granulation and cicatrization of the ulcer. It is preferably given an hour before meals, in order that it may readily reach the floor of the ulcer. If pain in the stomach is present, the bismuth may be combined with narcotics; as, for instance:

℞	Bismuth salicylate,	0.5	(7½ grains);
	Morphin hydrochlorate,	0.005	(1/12 grain);
	Extract of belladonna,	0.02	(⅓ ");
	Sugar,	0.03	(½ ").—M.

Make 10 such starch-capsules.
Dose: 1 capsule thrice daily before meals.

Should *hemorrhage from the stomach* take place, the patient should at once be put to bed. Small **bits of ice** may be given and be swallowed, an **ice-bag** applied to the epigastrium, about 8 minims of **fluid extract of ergot** with an equal amount of sterile water injected beneath the skin twice or thrice daily, and lead acetate administered internally; for instance:

℞ Lead acetate,	0.05 (¾ grain);
Powdered camphor,	0.02 (⅓ ");
Sugar,	0.5 (7½ grains).—M.

Make 10 such powders.
Dose: 1 powder thrice daily.

It is advisable to administer no food by the mouth for several days, substituting *nutrient enemata* of the composition already described. In case of *peritonitis* and *perforative peritonitis* rest, the application of an ice-bag to the abdomen, and administration of opium (0.03—½ grain; 1 powder every two hours) are necessary. Should collapse set in, 15 minims of camphorated oil should be injected beneath the skin every two or three hours. **Surgical intervention** has been practised successfully in a number of instances in the treatment of round ulcer of the stomach. Ulcers attended with uncontrollable and dangerous hemorrhage have been excised after exposure of the stomach, and in cases of perforative peritonitis the abdominal cavity has been opened, the pus removed as thoroughly as possible by sponging, and the rupture closed by suture. At times certain sequels of a round ulcer of the stomach necessitate operative intervention, as, for instance, cicatricial stenosis of the pylorus and hour-glass contraction of the stomach. For the relief of these conditions pyloroplasty and gastro-enterostomy have been practised with good results. In the *after-treatment* patients of means should be advised courses of treatment at the **springs**. An especial reputation in this connection is borne particularly by the springs of Carlsbad.

CARCINOMA OF THE STOMACH.

Etiology.—Carcinoma of the stomach, like carcinoma in general, is a disease of *advanced life*, usually developing after the fortieth year. Why this period of life is more predisposed to carcinoma is as yet unknown. Experience has shown that carcinoma of the stomach is *more common in men* than in women. It is also observed more commonly in some regions than in others, probably in consequence of local peculiarities in dietetic, injurious, and irritative influences acting upon the stomach. Without doubt **preceding disease of the stomach** has an influence upon the development of gastric carcinoma. Among such disorders are chronic gastric catarrh, round ulcer of the stomach, and irritation of the gastric mucous membrane by excessive indulgence in alcohol. **Injuries in the epigastrium** probably are not incorrectly looked upon as causes of carcinoma. The influence of *heredity*, however, is highly doubtful.

Anatomic Alterations.—Carcinoma of the stomach is almost always of *primary nature*; but few examples of secondary carcinoma of the stomach are on record. The most common *seat* is the pylorus, and next in frequency are the cardia and the lesser curvature. The fundus almost always escapes, even when the remainder of the stomach is involved in the new-growth. In some cases the tumor is circumscribed and attached to the gastric mucous membrane by a small base, and not rarely presenting a cup-shaped depression upon its surface. The shape of the tumor has under such conditions been compared with the appearance of a fungous growth, and on this account it has been designated a fungus of the stomach. In other instances diffuse carcinomatous infiltration takes place, which may attain a considerable extent, especially in the submucous connective tissue, and often sends medullary white strands of carcinomatous tissue into the connective-tissue septa of the muscular layer of the stomach, and even into the serosa. The wall of the stomach is often increased in thickness to several centimeters in the diseased area.

In accordance with the appearance of the neoplastic tissue, *several varieties of carcinoma of the stomach* are distinguished. If, in consequence of marked development of connective tissue, the new-growth is dense and deficient in fluid, it is designated a *fibrous carcinoma—scirrhus.* It is designated *medullary* or *alveolar carcinoma* when so-called "carcinoma-milk" can be scraped without difficulty from the carcinomatous tissue with a knife. *Colloid* or *gelatinous carcinoma* is attended with the presence of spaces filled with gelatinous contents resembling isinglass.

On microscopic examination the carcinomatous tumor is found to be constituted of epithelioid cells, which lie close together in a nest-like arrangement, and in consequence of which the individual collections of carcinoma-cells are separated from one another by connective-tissue septa. The carcinomatous tissue is developed as a result of abnormal proliferation of the chief cells of the lab-glands, the proliferation rapidly advancing into the submucous tissue, where it finds exceedingly favorable conditions for its further development. The portion of gastric mucous membrane involved in the new-growth frequently exhibits signs of chronic interglandular catarrh and even atrophy of the lab-glands (anadenia).

Carcinomatous tissue has a great tendency to undergo *disintegration.* Therefore the superficial layers of the new-growth are usually found destroyed, and thus the so-called *carcinomatous ulcer* is formed. In the process of disintegration *hemorrhage from or perforation of the stomach* may readily take place. The latter is often averted by the previous formation of adhesions between the stomach and adjacent organs in consequence of circumscribed peritonitis. Generally the *perigastric lymphatic glands* also undergo carcinomatous degeneration and are converted into hard, nodular tumors. Not rarely *secondary carcinoma develops in other organs,* most commonly in the liver.

Symptoms and Diagnosis.—The symptoms of carcinoma of the stomach are in part local, in part general. The latter consist in manifestations of *carcinomatous marasmus,* and these occur with especial readiness precisely in cases of carcinoma of the

℞ Lead acetate,	0.05 (¾ grain);
Powdered camphor,	0.02 (⅓ ");
Sugar,	0.5 (7½ grains).—M.

Make 10 such powders.
Dose: 1 powder thrice daily.

It is advisable to administer no food by the mouth for several days, substituting *nutrient enemata* of the composition already described. In case of *peritonitis* and *perforative peritonitis* rest, the application of an ice-bag to the abdomen, and administration of opium (0.03—½ grain; 1 powder every two hours) are necessary. Should collapse set in, 15 minims of camphorated oil should be injected beneath the skin every two or three hours. **Surgical intervention** has been practised successfully in a number of instances in the treatment of round ulcer of the stomach. Ulcers attended with uncontrollable and dangerous hemorrhage have been excised after exposure of the stomach, and in cases of perforative peritonitis the abdominal cavity has been opened, the pus removed as thoroughly as possible by sponging, and the rupture closed by suture. At times certain sequels of a round ulcer of the stomach necessitate operative intervention, as, for instance, cicatricial stenosis of the pylorus and hour-glass contraction of the stomach. For the relief of these conditions pyloroplasty and gastro-enterostomy have been practised with good results. In the *after-treatment* patients of means should be advised courses of treatment at the **springs**. An especial reputation in this connection is borne particularly by the springs of Carlsbad.

CARCINOMA OF THE STOMACH.

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Anatomic Alterations.—Carcinoma of the stomach is almost always of *primary nature*; but few examples of secondary carcinoma of the stomach are on record. The most common *seat* is the pylorus, and next in frequency are the cardia and the lesser curvature. The fundus almost always escapes, even when the remainder of the stomach is involved in the new-growth. In some cases the tumor is circumscribed and attached to the gastric mucous membrane by a small base, and not rarely presenting a cup-shaped depression upon its surface. The shape of the tumor has under such conditions been compared with the appearance of a fungous growth, and on this account it has been designated a fungus of the stomach. In other instances diffuse carcinomatous infiltration takes place, which may attain a considerable extent, especially in the submucous connective tissue, and often sends medullary white strands of carcinomatous tissue into the connective-tissue septa of the muscular layer of the stomach, and even into the serosa. The wall of the stomach is often increased in thickness to several centimeters in the diseased area.

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Symptoms and Diagnosis.—The symptoms of carcinoma of the stomach are in part local, in part general. The latter consist in manifestations of *carcinomatous marasmus*, and these occur with especial readiness precisely in cases of carcinoma of the stomach, because, in addition to the injurious influences usually exerted by carcinoma upon the general nutrition, in cases of carcinoma of the stomach the injurious effects of the disturbance of gastric digestion are superadded. As long as local symptoms are wanting the diagnosis will remain doubtful. Progressive emaciation, increasing pallor of the skin, and the occurrence of cachectic edema may, perhaps, arouse suspicion of latent internal carcinoma, but its situation in the stomach will be suggested only when gastric disturbances are at the same time present. Further, most extensive carcinoma of the stomach may exist in the absence of local symptoms referable to the stomach.

Among all of the *local manifestations* the demonstration of a *tumor of the stomach* is the most important from a diagnostic point of view. Usually this will be appreciable to the right of the median line just below the inferior margin of the liver, or it may extend from this situation toward the left in the course of the lesser curvature of the stomach. As, however, under normal conditions the pylorus, as well as the lesser curvature of the stomach, is concealed by the liver, it follows that tumors in the portions of the stomach named will become accessible only when they have attained considerable size and, by reason of their weight, are capable of dragging the stomach downward, so that as a result the pylorus and the lesser curvature of the stomach are directly applied to the abdominal wall. When the abdominal walls are thin and emaciated the gastric tumor is not rarely visible as a roundish and nodular mass. On palpation the growth will be felt to be of solid consistence and irregularly nodular. Almost always it is tender, even upon slight pressure. It is important that in contradistinction from new-growths of the liver and of the spleen displacement does not take place with the movements of respiration. Exceptions to this rule occur only when the tumor is adherent to the liver, and participates in its respiratory displacement; or, if the entire stomach is involved in carcinomatous degeneration, so that, by reason of the rigidity of its walls, it is no longer capable of yielding before the respiratory movement of the diaphragm. Further, care must be taken to avoid confounding the respiratory gliding of the abdominal walls over the tumor with the respiratory movement of the latter itself. Percussion over the gastric tumor yields a dull-tympanitic note.

Carcinomata of the cardia are never accessible to examination through the abdominal wall, but they can be detected only with the aid of the esophageal bougie, which encounters resistance 40 cm. (15¾ in.) from the edge of the teeth. The employment of the sound should never be omitted in any doubtful case of marasmus. Auscultation of the stomach is also important. If a patient with carcinomatous stenosis of the cardia be made to swallow fluid, there are heard, if the stethoscope is placed in the angle between the ensiform process and the left costal margin, either after a considerable length of time an injection-murmur and an expression-murmur, or after an unusually long interval but a single murmur. At the same time the murmurs are usually strikingly loud and long continued.

In the presence of gastric carcinoma the *functions of the stomach* are not altered in any such characteristic manner as to permit the recognition of the disorder with certainty. Frequently, almost constantly, the period of absorption, as determined by the potassium-iodid test, is prolonged, occupying, instead of ten or fifteen minutes, as much as from thirty to sixty minutes, or even more. The motor activity of the stomach also will be found diminished, and foreign bodies—for instance, cherry-pits—may remain in the stomach at times for months. Hydrochloric acid is almost always wanting in the gastric contents.

Boas has attached great diagnostic significance to the presence of lactic acid in the gastric contents, but without justification.

Persons suffering from gastric carcinoma complain principally of severe *epigastric pain* or *gastralgia*. This not rarely sets in toward night and disturbs sleep. Often there is *singultus* or *vomiting*. The regurgitated gases not rarely emit an offensive odor and that of hydrogen sulphid. The vomited matters often contain large numbers of sarcinæ, and long, curved bacilli, which have been considered responsible for the lactic-acid fermentation of the gastric contents. Of diagnostic importance is the occurrence of small masses of blood coloring-matter demonstrable microscopically.

At times portions of *carcinomatous tissue may be found in the vomited matters*, but this is an uncommon occurrence.

Most patients with carcinoma of the stomach complain of obstinate *loss of appetite—anorexia*. Thirst is not rarely increased. The tongue as a rule is coated. In diagnosis the presence of carcinomatous *induration and enlargement of the supraclavicular lymphatic glands* upon the left side is significant. The inguinal lymphatic glands, also, not rarely exhibit induration and enlargement. The *urine* is generally passed in small amounts; it is therefore usually deep red in color and concentrated, and as a rule contains much indican. The *bowels* are commonly constipated, although diarrhea may occur periodically. The *general nutrition* usually suffers early. The patients emaciate markedly within a short time, and acquire a pallid or waxy-yellow appearance. The latter is found on *examination of the blood* to be dependent upon diminution in the number of red blood-corpuscles and in the amount of hemoglobin.

If the anemia be advanced, *poikilocytosis* may be present. There may also be *hemorrhages into the retina*, *cutaneous edema*, and *small hemorrhages* and *foci of softening in the spinal cord*, such as take place in pernicious anemia.

Death occurs within a year, usually with progressive asthenia.

Among the *complications hemorrhage from the stomach* is the most conspicuous. This often gives rise to the *vomiting of blood—hematemesis*. Most frequently the vomited matters resemble coffee-grounds, or ink, or soot, while less commonly fresh blood is present. These variations are to be explained by the fact that, as a rule, small blood-vessels are opened in the process of disintegration of the carcinomatous tissue, so that the small amounts of blood extravasated remain for a considerable time in the stomach, while the hemoglobin undergoes alterations in consequence of the action of the acids of the gastric juice. Although hemorrhage from the stomach may be repeated frequently, it rarely causes death. Carcinoma at the pylorus frequently gives rise to *dilatation of the stomach—gastrectasis*. The pyloric ring may also be destroyed, and *pyloric incontinence* result. At times *secondary carcinoma develops in other organs*, and it may divert attention from the primarily involved stomach. In this connection secondary carcinoma of the liver especially should be mentioned. At times *rupture of the stomach* takes place, sometimes into the abdominal cavity, at other times, after previous adhesion of the stomach, into neighboring organs, rarely through the anterior abdominal wall. Carcinoma of the stomach is usually unattended with fever. At times, however, *febrile movement* occurs, and it may even be of intermittent type. Possibly it results from the absorption of toxic substances from the stomach. At times the patients unexpectedly become comatose, and die in *coma*. Such a result has been attributed to the absorption of toxic substances from the stomach; therefore an *auto-intoxication*. The urine frequently, though not constantly, exhibits a dark-red color on the addition of dilute solution of ferric chlorid—so-called ferric-chlorid reaction.

Prognosis.—The prognosis of carcinoma of the stomach is invariably unfavorable, as death results at the latest in the course of a few months.

Treatment.—No internal remedy is known that is capable of effecting a cure of carcinoma of the stomach. Attempts have been made to bring about this end by **operative measures.** Complete removal of the tumor by resection of the pylorus will be possible only when the carcinoma is not too extensive and is not bound by adhesions to adjacent organs, and has not yet given rise to metastases in other viscera, particularly not in the adjacent lymphatic glands. When signs of pyloric stenosis are present and resection of the pylorus is not possible, *gastro-enterostomy* should be effected; that is, the stomach should be sutured to a loop of small intestine, and at this point the lumen of the stomach and the bowel should be made continuous by means of an orifice of communication. In cases of carcinoma of the cardia *gastrostomy* may be attempted if the cardia is no longer permeable for food and drink, but this operation has not yet yielded brilliant results.

Internal treatment can only be purely symptomatic. The most important matter is that of **diet**. Such food should be prescribed as makes the smallest possible demands upon the digestive activity of the stomach; as, for instance, milk, milk with coffee, milk with tea, beef-soup with addition of peptone, milk with somatose, wine, old beer, rice-pap, mashed potatoes, etc. Wine of condurango may be prescribed to stimulate the appetite:

℞ Wine of condurango, 200.0 (6½ fluidounces).
Dose: 15 c.c. (1 tablespoonful) thrice daily.

The use of **dilute hydrochloric acid** (10 drops in a wine-glassful of tepid water 30 minutes after the midday and the evening meal) may be recommended. Morphin hydrochlorate will be required for the severe pain, although this should be prescribed only when absolutely necessary, as experience has shown that the dose must be progressively increased, as a result of which certain dangers may arise. Great relief is often afforded by systematic **irrigation of the stomach**, and under such treatment the bodily weight is frequently observed to increase considerably within a short time. By this means the stomach is freed from the stagnating remnants of food and also from toxic substances. From a diagnostic point of view it is further important to examine the irrigation-fluid for the presence of flakes that, upon microscopic examination, may prove to be carcinomatous tissue.

DILATATION OF THE STOMACH (GASTRECTASIS).

Etiology.—Dilatation of the stomach consists primarily in an *acquired increase in the capacity of the stomach*, although this is almost always invariably associated with *stasis* and *fermentation of the gastric contents*, so that the latter process must also be included in the clinical conception of gastrectasis. The most common cause for dilatation of the stomach is **stenosis of the pylorus**, and this is often due to cicatrized *ulcers*. *Carcinoma* of the pylorus may also give rise to stenosis of this orifice. **Benign hypertrophy of the pyloric ring** occurs less commonly, and obstruction of the pylorus by foreign bodies or gastric polypi still less commonly. At times the pylorus is obstructed by pressure or traction from without, as, for instance, by new-growths of the intestine, the omentum, the liver, or the kidneys, by wandering kidney, and the like. It will be easily understood that stenosis of the pylorus may readily give-rise to gastrectasis, for if the food stagnates in the stomach it causes stretching of this viscus by reason of its weight, and in addition there is the influence of fermentation of the gastric contents.

Also, in the absence of pyloric stenosis long-continued **over-filling of the stomach** with food may give rise to gastric dilatation. This condition is observed in gourmands and in diabetics, in whom the appetite is morbidly increased. The filling of the stomach with indigestible articles is also dangerous. For this reason conjurers suffer from gastrectasis in consequence of the practice of swallowing stones and other indigestible articles for the purpose of sustaining their supernatural powers. Finally, dilatation of the stomach is frequently a sequel of **diseases of the wall of the stomach,** as, for instance, *chronic gastric catarrh.* Gastrectasis has been observed also in connection with **extensive ulceration of the stomach.** At times **nervous influences** are operative by causing diminution in the tone of the gastric musculature. Anemic, nervous, and debilitated individuals (as, for instance, tuberculous patients), therefore, not rarely suffer from gastrectasis. It is noteworthy that **perigastric adhesions** may give rise to dilatation of the stomach. Gastrectasis is a most *common disorder,* which may appear even in children. Naturally it is often diagnosed when it does not exist, even by so-called "stomach-specialists," who do not deem it necessary to determine accurately the size of the stomach in every case.

Anatomic Alterations.—Dilatation of the stomach is characterized principally by the **increased size of the viscus,** the enlargement taking place in all directions. Upon opening the abdomen in advanced cases scarcely anything but the stomach comes into view, with its lesser curvature at the level of the ensiform cartilage and its greater curvature projecting into the true pelvis. Usually the enlarged organ contains a *large amount of fluid*—at times more than 20 liters; and if the wall of the stomach be palpated, not rarely the fluid can be observed to fluctuate to and fro. The **mucous membrane of the stomach** usually exhibits signs of a chronic catarrhal condition. The **walls of the stomach** at times present great thickening, especially of the muscular layer, which is often traversed by broad bands of connective tissue; at other times the walls of the stomach are unusually thin. Increased thickness of the walls of the stomach may be expected, particularly in association with stenosis of the pylorus, to a certain degree in the nature of compensatory muscular hypertrophy, while thinning attends conditions of general debility. A distinction has accordingly been made between *hypertrophic* and *atrophic gastrectasis.*

Symptoms and Diagnosis.—In order to demonstrate dilatation of the stomach the most convenient and most reliable method consists in **distending the viscus** artificially by means of **carbon dioxid.** For this purpose 1 or 2 teaspoonfuls of tartaric acid dissolved in a wine-glassful of water are swallowed, and immediately afterward a like amount of water containing 1 or 2 teaspoonfuls of sodium bicarbonate in solution. After the lapse of a few seconds the outlines of the stomach often become visible beneath the abdominal walls. On percussion or palpation with flexed fingers a sense of resistance like that yielded by an air-cushion will be perceived, which disappears below the greater curvature of the stomach. On percussion over the stomach a deep-tympanitic note is yielded, and on auscultation not rarely small moist râles are audible in the course of the greater curvature, resulting from ebullition of the carbon dioxid.

The distinctive feature of dilatation of the stomach consists in the **uniform enlargement of the viscus.** The greater curvature is not, as in health, situated above the umbilicus, but it is displaced for a greater or lesser distance below. Toward the left the stomach extends beyond the left anterior axillary line, and toward the right beyond the right parasternal line. Often the lesser curvature of the stomach, which in health is concealed by the liver, is displaced unduly downward, and is distinctly appreciable to the eye.

Displacement downward of the greater curvature of the stomach below the umbilicus, without displacement of the lateral boundaries of the stomach to the right and to the left, is not indicative of dilatation of the stomach, for such a condition may be due to *displacement downward of the stomach as a whole—gastroptosis.* Even a stomach enlarged in all directions is not necessarily to be considered as abnormally dilated, but the condition may be congenital—so-called *megalogastria.* In contradistinction from dilatation of the stomach, the functional activity of the organ is unimpaired when megalogastria exists.

In consequence of fermentative decomposition of the contents of the stomach the viscus is often so greatly distended with gas that its boundaries can be made out by inspection, palpation, and percussion, without artificial distention by means of carbon dioxid. If dilatation of the stomach be dependent upon pyloric constriction, active muscular waves may frequently be seen to pass from the cardia to the pylorus beneath the abdominal wall. At times bubbles of gas can be heard arising from the fermenting gastric contents. If the stomach be vigorously agitated, loud **splashing sounds** frequently occur, which some patients can induce by rapidly rising upon the feet, or by rapidly rolling from one side of the body to the other. These murmurs occur with especial frequency and loudness in association with dilatation of the stomach, but they appear also under all conditions in which the stomach contains at the same time both gas and liquid. At most it is distinctive of dilatation of the stomach that these sounds are appreciable when the abdominal wall below the umbilicus is suddenly jarred. With the physical alterations thus far described in cases of gastric dilatation manifestations indicative of **deranged functional activity of the stomach** are constantly associated. A peculiar variety of *vomiting* occurs with especial frequency. The patient may not vomit at all for several days in succession, but when vomiting occurs such enormous masses are expelled that the amount of vomited matter alone is indicative of dilatation of the stomach. The *vomited matter* is generally conspicuous for its markedly rancid or penetrating acid odor. If it be permitted to stand in a vessel in a warm room, it frequently continues to undergo fermentation, and eventually frothy matter may overflow the margin of the receptacle. Microscopic examination discloses, in addition to particles of food and bacteria, especially oblong yeast-cells, and layers of sarcinæ, at times also molds (Fig. 26). The vomitus is usually characterized by a highly acid reaction, although often it contains no free hydrochloric acid, whereas in consequence of fermentation of carbohydrates it contains an abundance of lactic, acetic, and butyric acids. The patient often complains of an acid, almost burning, taste on the part of the vomited matter, and the teeth are set on edge. Patients are frequently annoyed by *eructation—singultus.* After both vomiting and also eructation a burning, boring sensation not rarely remains behind the sternum—*heart-burn, pyrosis.*

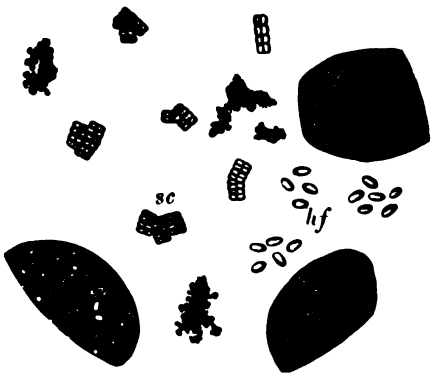

FIG. 26.—Yeast-cells and sarcinæ from the vomitus in a case of gastric dilatation; magnified 275 times (personal observation, Zurich clinic).

Eructation of combustible gases has been observed on a number of occasions, and is at times first noticed by patients on lighting a cigar. Analysis has shown that these gases consist, in addition to oxygen, nitrogen, hydrogen, and carbon dioxid, also of marsh-gas, carbon monoxid, oil-forming gas, and hydrogen sulphid.

The *functions of the stomach* are greatly altered in cases of gastric dilatation. The period required for the absorption of potassium iodid is prolonged (more than fifteen minutes). The motor activity of the stomach also has suffered considerable impairment. Free hydrochloric acid is frequently wanting in the gastric contents. The *appetite* at times suffers, as, for instance, in cases of carcinomatous stenosis of the pylorus. In other instances, on the contrary, the appetite is excessive, because but little of the food ingested is absorbed. There is almost always marked *increase in thirst*, as fluids likewise are absorbed only with difficulty by a dilated stomach, and also are but inadequately propelled into the intestinal canal. The *tongue* is frequently marked by unusual redness and cleanness, because it is irritated and cleaned by the highly acid vomited matters. The *urine* is usually passed in small amount. After abundant acid vomiting it not rarely yields an alkaline reaction, and a peculiar sediment, which, in addition to coffin-lid crystals of ammonio-magnesium phosphate, consists of rhombic plates of magnesium phosphate. The *bowels* are generally constipated. The *general nutrition* suffers in marked degree. The patients undergo great emaciation; their muscles become flabby, the skin thin like tissue-paper and deficient in fat, the face wrinkled, and the complexion ashy gray or grayish yellow. Some patients present an almost desiccated, mummy-like appearance. If the nutritive disturbance cannot be checked, death may result from progressive asthenia.

In some cases dilatation of the stomach is attended with peculiar *complications*, which have been attributed to *auto-intoxication*, upon the assumption that certain putrefactive alkaloids or ptomains have developed in the stomach, which exert an influence upon the nervous system especially. Under such conditions symptoms of *tetany* appear. This complication is not unattended with danger, and may be the immediate cause of death.

The *duration of dilatation of the stomach* will depend upon the fact whether the patients observe strict dietetic precautions or not. If they will obey appropriate medical advice, the disease may exist for decades, and be tolerated without serious discomfort. The course of events is often such that periods of aggravation and of improvement alternate accordingly as the mode of life is careful or indifferent.

Prognosis.—The prognosis of dilatation of the stomach depends, in the first place, upon the **causative factors.** It is from the outset unfavorable in cases of carcinomatous stenosis of the pylorus, for instance. If the condition is not dependent upon some disorder in itself dangerous to life, not only relief, but even cure, may be effected.

Treatment.—Dilatation of the stomach requires both **causal** and **dietetic treatment.** If the condition is dependent upon stenosis of the pylorus, an attempt should be made to correct this, and **surgical measures** will probably always be required for the purpose, as, for instance, resection of the pylorus—pyloroplasty, or bloodless dilatation of the pyloric ring. If the obstruction be irremediable, brilliant results are often attained by gastro-enterostomy; that is, the establishment of an artificial communication between the stomach and the bowel, and thus avoiding the pylorus. The suggestion of Bircher, to correct dilatation of the stomach by making a fold in the viscus, appears as yet to have been acted upon in but few instances. Especially good results may be attained by systematic **siphonage of the stomach,** in order to free the viscus from its excessive contents, and to afford it opportunity gradually to contract to its natural size.

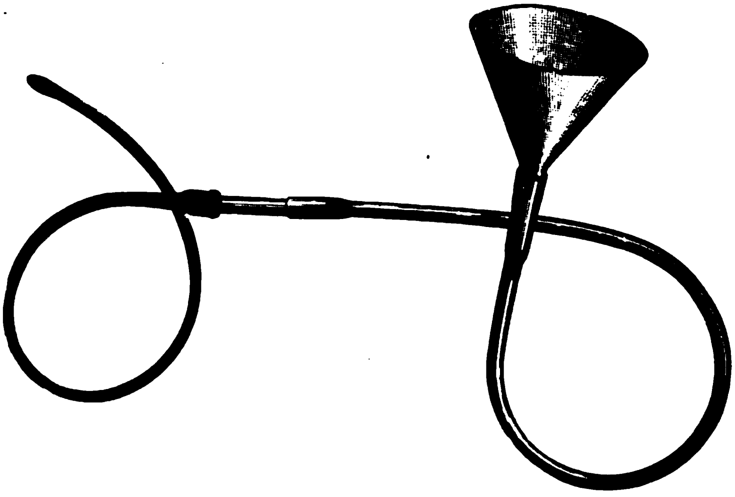

FIG. 27.—Stomach-siphon of soft rubber.

As a stomach-siphon a soft stomach-tube may be employed, and this is connected by a glass tube with a rubber tube 1 meter long. Into the free end of the rubber tube a glass funnel is introduced (Fig. 27). The stomach-tube is introduced as far as possible into the stomach, while the patient makes efforts at swallowing, and the warm water is permitted to flow into the funnel until the patient complains of a sense of fulness or no more water will enter. The rubber tube is then compressed between the fingers and the funnel is inverted downward into a vessel. If the pressure upon the tube be now relaxed, the water that has been introduced, together with the contents of the stomach, will escape. This procedure is repeated until the irrigating fluid returns perfectly clear. At times the discharge is prevented by obstruction of the fenestra of the tube by coarse particles of food, as, for instance, pieces of meat. Usually the obstruction can be overcome by strong pressure with the hand upon the tube or by the introduction of water into the funnel. Otherwise the tube must be removed and the lumen made clear. If the patient complain of marked acidity, it is well at the conclusion of the procedure to wash the stomach with artificial Carlsbad salt (one teaspoonful to a quart of tepid water). In order to prevent fermentation of food in the stomach, irrigation with *disinfectants* has also been practised. It is important to determine by means of graduates the amount of water introduced, and also that which escapes, for only if the latter be greater than the former can there be certainty that the contents of the stomach have been removed. Under the opposite conditions there is danger that the stomach would be more greatly overloaded. At first the irrigations are practised daily, and we make them, not, as some have advised, in the evening, but in the morning. Gradually one, then two, and finally several days are permitted to intervene. Patients learn how to irrigate the stomach in a short time, as a rule, and intelligent patients may be permitted without concern to practise the irrigation at home. Should signs of aggravation appear, the irrigation must again be practised with greater frequency. Most patients usually feel so much improved after the first irrigation that they can scarcely await the time for the next. The favorable influence of the procedure also becomes clearly appreciable in their appearance. The dull aspect again becomes fresher and animated. Bodily weight and vigor often increase with remarkable rapidity, and the complexion acquires a healthy color. Disagreeable accidents but rarely occur in connection with irrigation of the stomach. At times tetany has been observed to follow directly upon the manipulation, probably in consequence of the mechanical irritation of the stomach.

At the conclusion of the irrigation it is well for the patients to remain in bed for one or two hours, and to apply an **ice-bag to the epigastrium** in order to improve the tone of the musculature of the stomach. It may also be advantageous to practise **massage of the epigastrium,** or to apply the **faradic current,** or to inject **strychnin subcutaneously** (0.1 : 10 ; 0.3—4 minims).

Individuals in whom dilatation of the stomach has developed in consequence of over-eating should be provided with a rigid diet-list, so as to avoid overloading of the stomach in the future. In cases in anemic and nervous individuals **preparations of iron, courses of cold-water treatment,** and the like, are indicated. Relaxation of the abdominal walls requires the use of an **abdominal binder.** The **dietetic regulations** are the same in all varieties of dilatation of the stomach. Carbohydrates are to be avoided as strictly as possible, on account of the readiness with which they undergo decomposition. The ingestion of liquids should also be restricted. Patients should confine themselves especially to an animal diet, and the intervals between meals should not be too long. In order to relieve the stomach of catarrhal conditions of the mucous membrane that may be present, **courses of treatment at springs** are often of great utility, and particularly such as are followed at Carlsbad have yielded brilliant results.

DISPLACEMENTS OF THE STOMACH (DYSTOPIA VENTRICULI).

Displacements of the stomach are either *acquired* or *congenital. Congenital displacement of the stomach* occurs in connection with *transposition of the viscera,* when other organs are likewise displaced, so that, for instance, the heart may be situated on the right side of the thorax, the liver in the left hypochondrium, the spleen in the right, etc. Under such circumstances the cardia will be found upon the right and the pylorus upon the left side. This condition is unattended with other disturbances. *Acquired displacement of the stomach* is also known as *gastroptosis.* Attention has only of late been directed to the frequency of this occurrence. The condition is with especial frequency characterized by an abnormal depression of the stomach, so that when the viscus is distended with carbon dioxid the greater curvature will be found below the level of the umbilicus, while the transverse area appears rather diminished. There is often besides a wandering kidney on the right side, and the impression is created that both conditions are dependent upon the same cause, which most frequently consists in the pressure of constricting clothing. In addition, derangement in the functional activity of the stomach occurs, and with especial frequency anacidity of the gastric juice and motor weakness. The patients often complain of a sense of pressure or of pain in the epigastrium, of loss of appetite, eructation, and even vomiting. The wearing of constricting clothing should be interdicted and appropriate diet directed, and, in the presence of anacidity of the gastric juice, dilute hydrochloric acid prescribed.

GASTRIC NEUROSES.

Such disorders of gastric activity as are not attended with anatomic alterations in the wall of the stomach, but with *morbid activity of the gastric nervous supply*, are designated gastric neuroses. A distinction is made between *motor*, *sensory*, *secretory*, and *mixed gastric neuroses*, accordingly as the one or the other nerve-path is involved. At times the functional activity of the nerves is morbidly increased, at times diminished.

MOTOR GASTRIC NEUROSES.

NERVOUS VOMITING.

Symptoms, Diagnosis, and Prognosis.—As the name of the disorder indicates, nervous vomiting is characterized by more or less frequent **emesis.** This may be so abundant and occur with such constancy after each meal that the nutrition of the patient may become impaired to an alarming degree, with the possibility of starvation. In other instances the vomiting occurs only at certain times and transitorily. In contradistinction from the vomiting that occurs in connection with demonstrable anatomic disease of the stomach, it is peculiar to nervous vomiting that it usually takes place without preceding nausea, and that the matters ejected consist of the unaltered and unfermented contents of the stomach. The *duration of the affection* varies, and, together with the prognosis, is dependent upon whether the causative conditions can be removed or not.

Etiology.—Nervous vomiting is not rarely due to **diseases of the brain and the spinal cord** (concussion of the brain, cerebral tumor, abscess of the brain, cerebral hemorrhage, parasites, cerebral meningitis, tabes dorsalis, etc.). It has been observed also in association with central neuroses, and with especial frequency with hysteria. Often nervous vomiting is of **reflex origin.** The best-known instance of this kind is the vomiting of pregnancy. Frequently nervous vomiting attends biliary and renal calculi. Irritative conditions in the most diverse organs may, however, also give rise to nervous vomiting. Vomiting dependent upon **toxic influences** may likewise be considered of nervous origin. This may be induced intentionally by certain remedies (emetics, such as ipecacuanha-root, tartar emetic, copper sulphate, apomorphin hydrochlorate); or it may be due to certain poisons generated within the body itself as a result of metabolic processes (autointoxication), as, for instance, in uremia and cholemia. Nervous vomiting occurs most commonly in *women*, because it is often dependent upon disease of the uterus and its appendages. It is less commonly encountered in children than in adults, as many of the causative conditions are observed only in the latter.

Treatment.—Nervous vomiting requires, in the first place, **causal treatment.** Symptomatically, **sulphuric ether** (5 drops on sugar repeatedly), **ethereal tincture of valerian** (25 drops thrice daily), **tincture of iodin** (1.0 : 200; 15 c.c.—1 tablespoonful—every two hours), and **morphin hydrochlorate** (subcutaneously) are indicated. The swallowing of small bits of ice is also advisable.

NERVOUS ERUCTATION.

Nervous eructation occurs especially in cases of *hysteria*, *neurasthenia*, and *hypochondriasis*. The patients suffer from eructation of gas that they have swallowed a short time previously and often expel with a loud, belching sound. At times the condition may persist for hours or even days, or it may occur in paroxysms in consequence of psychic disturbances. Sometimes an attack can be induced by pressure upon certain parts of the body. The disorder is unattended with danger.

The **treatment** should be partly psychic and in part directed against the primary affection.

NERVOUS REGURGITATION.

Regurgitation of food is a phenomenon that occurs also in healthy persons when the stomach is irritated by the ingestion of an excess of food or of food that is too cold or too hot, or by too rapid eating and insufficiently masticated food. The condition may be morbidly exaggerated in cases of *hysteria*, *neurasthenia*, and *hypochondriasis*. If fermentative processes in the stomach are superadded, the regurgitated matters may have an acid or a rancid taste. Regulation of the diet, the coöperation of the patient in an effort to suppress the regurgitation, and treatment of the primary disorder are capable of correcting the condition.

PERISTALTIC UNREST OF THE STOMACH (NERVOUS TORMINA OF THE STOMACH).

Nervous and neurasthenic individuals complain at times of a *disagreeable feeling in the stomach* and of *restless movements in the epigastrium,* which not rarely set in during the evening and even during the night, and disturb sleep. These manifestations are frequently associated with visible active contractions of the musculature of the stomach, and peristaltic movements may be observed passing slowly from the cardia to the pylorus. Care must be taken to avoid confounding the condition with the active movements of the stomach that occur in the presence of pyloric stenosis in the endeavor to overcome the obstruction to the passage of the chyme from the stomach into the duodenum. The absence of the causes and of the alterations of stenosis in the pyloric region, and the normal character of gastric digestion in other respects, are indicative of nervous unrest of the stomach.

Treatment should be directed against the primary disorder. In addition, the diet should be regulated. Rest in bed and hot cataplasms should further be advised.

HYPERMOTILITY OF THE STOMACH.

Hypermotility of the stomach may be recognized from the fact that after a meal of meat the stomach is empty in a short while, at times as early as an hour. The patients complain of excessive appetite.

TONIC SPASM OF THE MUSCULATURE OF THE STOMACH.

Spasm of the cardia—cardiospasm—prevents the unobstructed entrance of food into the stomach. The stomach-tube also encounters resistance at the cardia—thus at 40 cm. ($15\frac{3}{4}$ inches) beyond the margin of the teeth; but this frequently yields, in contradistinction from organic stenosis of the cardia, if the sound be permitted to remain quietly for a few seconds. The disorder occurs in nervous persons, but may also be induced by reflex influences in connection with some diseases of the stomach, as, for instance, gastric ulcer.

Spasm of the pylorus causes obstruction to the passage of food from the stomach into the intestine, and may be readily confounded with organic stenosis of the pylorus. At times there is spasm simultaneously at the pylorus and at the cardia. Under such conditions marked distention of the stomach with gas may take place, and this may give rise to disagreeable sensations in the epigastrium and to conditions of dyspnea—so-called dyspeptic asthma. The distinctive feature of pure nervous pyloric stenosis is the paroxysmal occurrence of the phenomena mentioned.

Tonic spasm of the entire stomach occurs in connection with pyloric stenosis, and also as an independent neurosis. The stomach appears contracted with board-like hardness. Often painful sensations in the epigastrium are appreciable during the spasmodic attack.

All spasmodic conditions of the stomach should be treated by dietetic measures, hot cataplasms, bromids, or injections of morphin. Besides, the primary disorder should be relieved.

INSUFFICIENCY OR INCONTINENCE OF THE PYLORUS.

Insufficiency of the pylorus is characterized, on generation of carbon dioxid in the stomach, by failure of distention to take place or by its transitory occurrence; whereas protrusion of the entire abdomen occurs because the gas has passed into the intestine from the stomach through the inefficient pylorus. The condition is encountered in cases of hysteria, neurasthenia, and spinal softening, and as a purely nervous disorder, although it may result in connection with severe gastric catarrh and destruction of the pyloric sphincter by ulceration or carcinoma. Physiologically incontinence of the pylorus is believed to be present when the stomach is empty.

The **treatment** should be directed against the primary disorder.

RUMINATION (MERYCISM).

Individuals who practise rumination return the food from the stomach into the mouth a short time after its ingestion, often rechewing it with great satisfaction, and many experiencing a more agreeable sensation than when the food was originally ingested. Some patients repeat the practice after each meal; others only when they eat hastily and do not masticate the food sufficiently. The nutrition may remain unchanged; and only when, in consequence of gastric catarrh or dilatation of the stomach, decomposition of the food takes place, and the patients, by reason of the disagreeable taste of the food, eject the regurgitated material, do emaciation and anemia occur. The functional activity of the stomach varies, and at times anacidity and at other times hyperacidity of the gastric juice have been observed. Rumination is a much more common disorder than is generally believed. I have quite accidentally detected a number of persons engaged in rumination who from a sense of shame were cleverly able to conceal the unpleasant practice. Among the causative factors *nervous diseases* must be considered, as, for instance, chorea, epilepsy, and idiocy. Individuals suffering from *pulmonary tuberculosis* not rarely practise rumination. *Imitation* is an important etiologic factor. *Dietetic errors* are often operative, particularly too hurried eating and insufficient mastication of the food. At times rumination develops in the course of *diseases of the stomach and the intestines* (a blow upon the stomach, constipation). Patients frequently state that the disorder has begun with eructation, that subsequently they acquired the ability of inducing the eructation intentionally, and that finally they became ruminants, the contents of the stomach being forced into the mouth unconsciously. Many are able to suppress the practice voluntarily. Anatomic alterations of a distinctive character are unknown. The presence of dilatation at the cardia, constituting a so-called fore-stomach, is an accidental and rare condition. For the development of rumination weakness of the musculature at the cardia is probably necessary, as otherwise the food could not be returned.

In the **treatment** the coöperation of the patient and his willingness to suppress the practice are the most important factors. Some nervous, however, are unwilling to be deprived of the pleasure afforded by rumination. It is important, further, that the food should be eaten slowly, and be thoroughly masticated. At times rumination can be prevented by the swallowing of bits of ice. Besides, nervines, as, for instance, bromids, should be employed.

ATONY OF THE STOMACH.

Atony of the stomach consists in diminution in the tone of the [illegible] musculature and in motor weakness. The condition is associated with the danger that the food may remain stagnant in the stomach for a long time, and undergo decomposition, with [illegible] of dilatation of the stomach. The patients complain [illegible] of a sense of fulness and of pressure in the epigastrium. The condition is frequently encountered in anemic, [illegible], nervous, hysterical, and hypochondriacal individuals. [illegible] curable. A nutritious diet, consisting rather of [illegible] liquid food, should be prescribed, and the general [illegible] of the body promoted by cold frictions and massage. [illegible] effort should be made to correct the primary disorder.

SENSORY NEUROSES OF THE STOMACH.

NERVOUS GASTRALGIA.

Symptoms and Diagnosis.—Nervous gastralgia is often [illegible] intense pain in the epigastrium, which appears at [illegible] the stomach is empty, and at other times soon after [illegible] food, in the latter event particularly when food or [illegible] that is not well borne even in a condition of health. [illegible] attack of gastralgia has been preceded by psychic [illegible] in women it may occur at the menstrual period. [illegible] the pain is frequently so great that, in consequence [illegible] of the cutaneous vessels, the skin becomes pale [illegible] covered with a clammy sweat. The pulse becomes [illegible] moan, and double themselves up. Syncope [illegible] contractions may occur. At times the dis- [illegible] with a single attack of pain or after a small number, while in other instances it may persist for months and years. In the latter event several attacks may occur in the course of a single day. Examination of the stomach yields little information, and it is often difficult to differentiate nervous gastralgia from that attendant upon anatomic disease of the stomach, as, for instance, round ulcer of the stomach. It is noteworthy that nervous gastralgia is at times relieved by firm pressure in the epigastrium, so that not rarely during the attack the patients make such pressure with their hands or press the epigastrium against some resistant body. If the ingestion of food is not observed to exert any influence upon the occurrence of an attack of pain, this is rather indicative of nervous gastralgia. It is further contended, also, that nervous gastralgia disappears upon galvanization of the epigastrium, with the positive pole (anode) applied.

Prognosis.—Nervous gastralgia is a most distressing, but not a fatal disease. It, therefore, occasions no concern with regard to the continuance of life.

Etiology.—The disorder occurs most commonly in association with certain **central neuroses,** particularly hysteria and neurasthenia. **Anatomically demonstrable disease of the nervous system** may also be attended with gastralgia, as, for instance, tabes dorsalis. The disorder is often dependent upon **reflex influences.** Women with disease of the uterus or the ovaries especially suffer not rarely from nervous gastralgia. In some families the disease is transmitted by **heredity,** probably because the morbid condition of the nervous system is inherited. Mental over-exertion, wasting discharges, excessive indulgence in alcohol, tea, and tobacco, and sexual excesses engender an undeniable predisposition to the disease. *Women* are most commonly attacked because a large number of the causative influences are operative among them. Children are attacked much less commonly than *adults.*

Treatment.—In the treatment of nervous gastralgia two indications should be borne in mind: in the first place, the relief of the individual attack of pain, and in addition the prevention of recurrence—**symptomatic** and **causal treatment.** In the attack of pain *hot cataplasms* may be applied. If the pain be intense, a *subcutaneous injection of morphin* should be given, but the patient should never be entrusted with a solution of morphin and a syringe for use at his discretion, as the danger of abuse and of addiction to the drug is too great. *Causal treatment* should be directed against the primary disorder, and will be based upon the principles applicable to the individual diseases.

DISORDERS OF THE SENSE OF HUNGER AND OF SATIETY (ACORIA; BULIMIA; PICA).

Disorders of the sense of hunger occur, like most other gastric neuroses, especially in cases of *hysteria* and *neurasthenia*, and they disappear, as a rule, when the primary disorder is cured. *Acoria* is a condition in which the sense of hunger is lost, so that the patients do not realize when they should take food. In other persons the sense of satiety is lost, and they are exposed to the danger of overloading the stomach. *Bulimia* or *excessive hunger* is characterized by a morbidly ravenous appetite, so that the patient can scarcely await the usual hour for meals, and often endeavors to suppress the sense of hunger by means of chocolate and other confections, which he constantly carries about with him. *Pica* is an unnatural desire for unusual and at times indigestible articles of food, as, for instance, ink, slate-pencils, vinegar, and the like.

SECRETORY NEUROSES OF THE STOMACH.

HYPERCHLORHYDRIA.

Hyperchlorhydria, less accurately designated also hyperacidity or superacidity, indicates an increase in the hydrochloric acid of the gastric juice, so that the total acidity of the gastric contents after a trial-breakfast may exceed 70, and the percentage of free hydrochloric acid may exceed 0.25. The patients complain principally of a disagreeable sense of pressure and of burning in the epigastrium. The condition at times accompanies a number of diseases of the stomach, as, for instance, round ulcer of the stomach; and it occurs also as an independent neurosis, particularly in neurasthenics. In the latter event efforts should be directed to the relief of the primary disorder. In addition, systematic irrigation of the stomach with Carlsbad salt (1 teaspoonful dissolved in 1 quart of tepid water) should be practised, and a generous animal diet, including but little amylacea, be permitted, as the digestion of starch is particularly interfered with by the excess of free hydrochloric acid.

ANACHLORHYDRIA AND HYPOCHLORHYDRIA.

Anachlorhydria and hypochlorhydria are known also as anacidity and hypacidity or subacidity, and are characterized by either total absence of free hydrochloric acid from the gastric contents or its presence in amounts below 0.15 per cent. The total acidity may [illegible]. These conditions occur in con[illegible] diseases of the stomach (acute and chronic [illegible] amyloid degeneration of the [illegible] extensive cicatrix-formation), but they [illegible] neuroses of the stomach. I have [illegible] frequency in association with a wandering right kidney and gastroptosis. They are encountered also, as a rule, in the presence of fever and of anemic states.

The **treatment** consists in the administration of dilute hydrochloric acid (10 drops in a wine-glassful of tepid water half an hour after meals), and in the correction of the primary disorder.

HYPERSECRETION OF THE GASTRIC JUICE (GASTROSUCCORRHEA).

Hypersecretion of the gastric juice, also known as gastrosuccorrhea, is characterized by the presence in the stomach, even in the morning before food is taken, of as much as several hundred cubic centimeters of fluid, which, from the amount of free hydrochloric acid that it contains, and its ability to digest albumin, is without doubt gastric juice. The disorder may be associated with hyperchlorhydria, and particularly under such conditions the patients usually complain during the night of a sense of boring and of burning in the epigastrium, and of increased thirst. The affection may be persistent or occur periodically, so that a distinction must be made between *continuous* and *paroxysmal gastrosuccorrhea.* Relief can be afforded by systematic irrigation of the stomach with a lukewarm solution of Carlsbad salt (1 teaspoonful to 1 quart of tepid water), and by the use of animal food, particularly at night. In addition, the nervousness usually present will require treatment.

MIXED NEUROSES OF THE STOMACH.

NERVOUS DYSPEPSIA.

Symptoms, Diagnosis, and Prognosis.—Dyspepsia signifies primarily only embarrassed gastric digestion. This manifests itself, in cases of nervous dyspepsia, particularly by the development of various discomforts after the ingestion of food. A sense of pressure in the epigastrium, abdominal distention, and frequent eructation are complained of with especial frequency. In addition there occur general nervous disturbances, particularly a sense of pressure in the head, mental confusion, vertigo, tinnitus aurium, a sense of fear, palpitation of the heart, redness of the skin, sweating, and the like. Examination of the functional activity of the stomach frequently discloses the existence of anachlorhydria or hypochlorhydria and diminution in the motor activity of the stomach, while, on the other hand, all signs indicative of anatomic alteration in the stomach are wanting. The patients are generally conspicuous on account of their pallid appearance, and frequently also by reason of their emaciation. In addition, they usually exhibit evidences of neurasthenia or hysteria. The disorder is troublesome and obstinate, but the prognosis is good, as it is scarcely capable of causing death.

Etiology.—Nervous dyspepsia is a common result of neurasthenia, hysteria, and hypochondriasis. As modern methods of living are highly conducive to the development of neurasthenia, it should not occasion surprise that nervous dyspepsia is a common disease of the stomach.

Treatment.—Nervous dyspepsia will be most successfully treated if attention is directed primarily toward the general nervous condition. In addition, a nutritious but easily digestible diet should be prescribed. It is noteworthy that the patients, as a rule, bear badly courses of treatment at the springs, such as are frequently directed by physicians who have attributed the symptoms of nervous dyspepsia to anatomic alterations in the stomach.

Under the name of *periodic vomiting* v. Leyden has described attacks of vomiting, associated with gastralgia, occurring in anemic and nervous individuals. Rossbach has designated as *gastroxynsis* attacks of excessive secretion of hydrochloric and lactic acids in the stomach, giving rise to vomiting, headache, and vertigo. Nervous individuals and heavy smokers especially are attacked. The condition can be relieved by irrigation of the stomach. The attacks not rarely can be controlled by the ingestion of hot water.

V. DISEASES OF THE INTESTINES.

ACUTE INTESTINAL CATARRH.

Etiology.—Like acute gastric catarrh, acute intestinal catarrh also develops most commonly as a result of dietetic errors, and frequently both disorders exist together. Explanation is scarcely required for the fact that if the contents of the stomach are abnormally irritating the intestines also will be involved in the morbid process as soon as it has taken up the irritating gastric contents. The nature of the dietetic error may be most varied. *Excess in food and drink* may readily become injurious from the occurrence of fermentation in the ingested matters in the digestive tract, with irritation of the mucous membrane. Acute intestinal catarrh develops still more readily after the *ingestion of decomposed food and drink*, as, for instance, after the eating of unripe fruit or vegetables, fermented drinks, putrid water, etc. In this category belongs also the acute intestinal catarrh of children, which occurs with especial frequency in the hot months of summer, and is dependent upon fermentation of the milk ingested. Also, the ingestion of *food that is too hot or too cold*, or is insufficiently masticated, or is indigestible, is capable of inducing acute intestinal catarrh. Some persons exhibit an idiosyncrasy to certain kinds of food, as, for instance, cucumbers, prunes; that is, they develop symptoms of acute intestinal catarrh regularly after the ingestion of certain articles of food, even when these are taken in the smallest amounts.

Acute intestinal catarrh of toxic origin develops after the swallowing of certain irritant poisons (acids, alkalies, arsenic, mercuric chlorid), but this is not a common mode of origin. Acute intestinal catarrh may be caused also by excessive use of purgatives. There is no doubt that *refrigeratory* (*rheumatic*) *intestinal catarrh* resulting from exposure to cold occurs. Thus, a cold bath when the body is in a state of perspiration may give rise to acute intestinal catarrh; sleeping upon damp ground has also at times been found to be a cause. I have in several instances observed acute intestinal catarrh to occur after the application of an ice-bag to the abdomen, so that it became necessary to withhold the application. Scarcely anything is known definitely with regard to *traumatic intestinal catarrh* in so far as external injuries are concerned. On the other hand, acute intestinal catarrh may result from internal injuries, as, for instance, in connection with constipation, in consequence of the irritation due to hardened scybalous masses, after the swallowing of bones or fruit-stones, and from the presence of biliary calculi in the bowel. Possibly some cases of acute intestinal catarrh associated with the presence of worms in the intestine should be included in this category. *Epidemic acute intestinal catarrh* occurs at times in the hot months of summer or autumn as an independent disorder. It may be so severe as to suggest the clinical picture of Asiatic cholera, and it has then been designated also cholera nostras s. europæa. *Secondary acute intestinal catarrh* may either arise by extension from adjacent disease (peritonitis) or accompany other intestinal or constitutional disease (typhoid fever, dysentery, cholera, carcinoma of the intestine, invagination of the intestine, febrile infectious diseases, etc.). Acute intestinal catarrh occurs at all periods of life. It attains a wide distribution in infancy on account of the readiness with which milk undergoes fermentation, and annually many thousands of children die from acute gastro-enteritis—cholera infantum.

Anatomic Alterations.—An inflamed intestinal mucous membrane, like other inflamed mucous membranes, exhibits **redness, swelling,** and **increased secretion.** The redness, which results from distention of the vessels of the mucous membrane with blood, is usually most pronounced upon the summit of the villi and the prominences of the folds. Here and there, probably, some blood-vessels have ruptured and **small hemorrhages** have taken place. The swelling of the mucous membrane is appreciable in the increased thickness of the tissues and excessive infiltration with blood-plasma. The **lymph-follicles** are involved in the swelling with especial regularity. They are increased in size, and are usually surrounded by a zone of hyperemic vessels. At first the swelling of the lymph-follicles depends only upon serous infiltration. They therefore resemble watery vesicles, and on puncture they give exit to a drop of clear fluid, while they collapse at the same time. Later, on the other hand, inflammatory hyperplasia of lymph-cells takes place in them, and they then acquire an opaque appearance and no longer collapse when punctured. The increased secretion of the mucous membrane is manifested by the accumulation upon the surface of the membrane of vitreous mucus, or mucus presenting a greenish-gray mottling in consequence of admixture with exfoliated epithelial cells or with pus. If the inflammation of the mucous membrane be intense, it will extend to the serosa, which then appears markedly hyperemic and of a rose tint. The **contents of the intestine** are usually thin and fluid. Not rarely they consist of a flocculent admixture of exfoliated epithelial cells from the intestinal mucous membrane. If the intestinal evacuations have been numerous and copious, the contents of the bowel may be gray and deficient in bile. In connection with severe intestinal catarrh the **mesenteric lymphatic glands** may also be involved and present swelling and redness. Acute intestinal catarrh may involve the entire bowel or only certain sections; and in the latter event, in accordance with the seat of the disease, a distinction has been made between acute duodenitis, jejunitis, ileitis, typhlitis (catarrh of the cecum), appendicitis (catarrh of the vermiform appendix), colitis, and proctitis (catarrh of the rectum). Ileocolitis is most commonly encountered. It is worthy of note that not rarely the most intense manifestations during life are attended with only slight alterations in the intestinal mucous membrane, obviously because a portion of the anatomic changes has disappeared in the cadaver.

Among the *complications* of acute intestinal catarrh superficial loss of tissue, *erosions of the bowel, more profound ulceration of the mucous membrane,* and *destruction of the lymph-follicles of the intestine* may be mentioned.

Symptoms and Diagnosis.—The symptoms of acute intestinal catarrh vary in accordance with the extent of the disease and with the portion of the bowel involved. If the large intestine is the seat of the disease, diarrhea occurs, in consequence of the increased peristaltic activity; the contents of the bowel are so rapidly sent through the large intestine that sufficient time is not allowed for inspissation to take place. Catarrhal conditions confined to the small intestine, on the other hand, are unattended with diarrhea, for the fecal matter acquires its usual consistency in the uninjured large intestine.

In the most common variety of acute intestinal catarrh—*ileocolitis*—there occur, at first, in many cases **rumbling and noises in the abdomen—borborygmi**—which result from unusually active and vigorous intestinal movement and the associated rapid propulsion of the intestinal contents. In consequence of decomposition of the intestinal contents there is active development of gas in the bowel, and the abdomen becomes distended—**intestinal meteorism** or **tympanites.** The gas seeks a means of exit downward, and frequently **flatus** occurs, and this is often attended with an offensive, almost fetid odor. Often there is **frequent desire for stool, and the stools** themselves are thin, not rarely like water. The dejections are frequently marked by a decomposing or putrid odor, and they vary between brown, yellow, green, and gray in color. At times the stools contain unusually large amounts of undigested food (meat-fibers, tendon, portions of vegetables)—so-called lientery. Of especial significance in diagnosis is the presence of mucus in the stools. The appearance of blood is one of the less common phenomena. The reaction of the stools is usually acid. Frequently the evacuated matters are abundantly admixed with gas, and are frothy. Microscopic examination of the feces discloses, in addition to numerous remains of food, desquamated epithelial cells, round cells, and crystals of cholesterin, fat at times, also Charcot-Neumann crystals (pointed double pyramids), and, besides, numerous bacteria, although it has not yet been possible to isolate specific bacteria. Yeast-cells also are frequently found in the stools. The number of bowel-movements is subject to great variation (from two to twenty in twenty-four hours). At times the patients are scarcely able to leave the commode, from a fear that they will at once again be compelled to resort to it, and that they may fail to reach it in time. The desire for stool appears with especial frequency after the ingestion of food. It is frequently preceded by **abdominal pain—tormina**; or there is escape of gas after pain has first appeared in the abdomen, and this likewise changes its position with the onward movement of the gas. If gastric catarrh exist in addition to intestinal catarrh, there will be present **nausea, vomiting, loss of appetite,** and **coated tongue.** Often there is **labial herpes.**

Acute intestinal catarrh may be unattended with alteration in the **bodily temperature,** while in other instances, on the contrary, febrile movement is observed. Many patients complain of general languor and malaise, and of drawing pains in the muscles and joints. **Thirst** is usually increased under all conditions, in consequence of the loss of fluid with the loose bowel-movements. The **abdomen** is frequently distended and tender to touch. The **urine** is usually voided in diminished amount. It is concentrated, and often deposits a sediment of urates. The amount of indican is frequently increased. Not rarely there is slight albuminuria, with cylindruria. Severe diarrhea causes a sense of weakness, and may even give rise to manifestations of collapse. As in Asiatic cholera, there may also be pains in the muscles, especially in those of the calves, and the voice may be high and hoarse.

The *duration of acute ileocolitis* varies from a few days to between two and four weeks. In adults recovery is the rule.

Acute catarrhal duodenitis can be recognized only when the choledoch duct also is involved in the morbid process. This may result from obstruction at the orifice of this duct in consequence of the swelling of the duodenal mucous membrane; or from the entrance of a plug of mucus from the intestinal mucous membrane into the duct and causing its occlusion; or, finally, from thickening of the mucous membrane of the duct in consequence of participation in the inflammatory process. Under all of these conditions biliary stasis and *icterus* develop, and the condition has then been designated gastroduodenal icterus, or catarrhal jaundice.

Jejunitis and *ileitis* cannot be diagnosed with certainty.

Typhlitis and *appendicitis* will be considered in a special section.

Proctitis is characterized by persistent desire for stool and pain when the bowels are moved—*anal tenesmus*. The anal sphincter is usually contracted spasmodically. Digital examination of the rectum causes severe pain. The mucous membrane of the rectum seems hot and unusually relaxed. Considerable mucus remains adherent to the finger, which at times is streaked and dotted with blood. After the condition has existed for some time paralysis of the anal sphincter sometimes develops, so that thin fecal matter escapes continuously from the rectum, moistens and irritates the skin in the anal region, and excites inflammatory reaction. There may also be prolapse of the bowel.

Prognosis.—The prognosis of acute intestinal catarrh is favorable if the disease occurs in adults, and no serious primary disorder is present. In infants acute intestinal catarrh is quite a serious disease, and in the aged also there is danger that death may result from asthenia.

Treatment.—Prophylactic measures may be extremely useful. **Care in diet** particularly should be observed. The treatment of acute intestinal catarrh depends upon the causative factors—**causal therapy**—and in part also upon the seat of the disease. In all varieties of the disease a febrile patient with frequent bowel-movements should remain in bed, keep a hot poultice applied constantly to the abdomen, and avoid solid food for several days. Weak tea, coffee, water with red wine, mucilaginous soups, and almond-milk may be permitted. Should intestinal catarrh be dependent upon dietetic errors, it is often most advantageous to prescribe a **purgative** (castor-oil, 25 c.c.—6 teaspoonfuls—in a cup of coffee; or calomel, 0.5—7½ grains), in order to expel speedily the fermenting and irritating intestinal contents. Often the symptoms disappear as soon as the bowels are emptied. Should the diarrhea persist, and frequent rumbling indicate abnormal increase in intestinal peristalsis, **preparations of opium** should be prescribed in order to keep the bowels quiet.

℞ Powdered opium,	0.03 (½ grain);
Sugar,	0.5 (7½ grains).—M.

Make 10 such powders

℞ Powder of ipecacuanha and opium, 0.3 (4½ grains);
Sugar, 0.5 (7½ ").—M.
Make 10 such powders.
Dose: 1 powder every three hours.

℞ Tincture of opium,
Ethereal tincture of valerian, each, 10.0 (2½ fluidrams).—M.
Dose: 20 drops thrice daily upon sugar.

℞ Morphin hydrochlorate, 0.3 (4½ grains);
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: From 0.25 to 0.5 (4 to 8 minims) subcutaneously.

It is often useful to combine preparations of opium with *astringents* (*styptics*); a combination of bismuth salicylate with opium is particularly reliable:

℞ Bismuth salicylate, 0.5 (7½ grains);
Powdered opium, 0.03 (½ grain);
Sugar, 0.3 (4½ grains).—M.
Make 10 such powders.
Dose: 1 powder every three hours.

Duodenitis and *gastroduodenal catarrh* should be treated according to the rules that have been laid down for the treatment of acute gastric catarrh. If jaundice be present, special care should be taken that all fatty substances are excluded from the diet. *Acute inflammation of the rectum* is readily accessible to local treatment, particularly with irrigation of the bowel (infusion) and suppositories. The latter, containing narcotics, may be employed especially when tenesmus is severe; for instance:

℞ Morphin hydrochlorate, 0.03 (½ grain);
Cocoa-butter, sufficient to make 3 suppositories.
Dose: 1 suppository twice or thrice daily.

ACUTE GASTRO-INTESTINAL CATARRH IN INFANTS.

Etiology.—Infants are in high degree predisposed to acute gastro-intestinal catarrh, and a large number of children succumb annually to this disease. In the first place, milk is a readily decomposed article of food, and, besides, the gastro-intestinal mucous membrane of infants appears particularly sensitive. Experience has shown that infants given mother's or wet-nurse's milk suffer much less commonly than those nourished with **cow's milk** or **substitutes for milk.** Milk undergoes decomposition the more readily the higher the external temperature, and in accordance with this fact acute gastro-intestinal catarrh occurs with especial frequency in the hot months of summer and autumn (from July to September). The condition has, therefore, also been designated *summer diarrhea of infants.* The greater the want of cleanliness in the dairies from which the milk is obtained, the greater is the danger of fermentative bacteria gaining entrance into the milk, especially with small particles of excrement from the cow, so that the milk is contaminated from the outset. It is important that the milk be thoroughly boiled, in order, so far as possible, to destroy fermentative bacteria; that the receptacles for the milk be kept cold and covered; that remnants of milk not drunk by the infant be thrown away or be used for other purposes; and that the bottles and the nipples be kept scrupulously clean. Failure to observe these precautions may expose the infant to grave danger. At times error is committed in giving the infant milk in too large quantities or at irregular intervals. Experience has shown that infants bear poorly the milk of cows supplied with **green fodder.** **Dentition** undoubtedly predisposes to gastro-intestinal catarrh. It is not rarely observed that a perfectly healthy child is attacked by diarrhea, and in spite of the greatest care in feeding, only when a new tooth is in process of irruption—so-called *dentition-diarrhea.* The circumstance that the connection between the two conditions is not recognized cannot justify doubt of the fact itself. Some infants are attacked by severe acute gastro-intestinal catarrh when the attempt is made to wean them from an exclusive milk-diet—*diarrhœa ablactatorum;* and at times the disease cannot be controlled until the exclusive milk-diet is resumed. The sensitiveness of the infant's stomach and intestines with regard to a milk-diet may be recognized from the fact that some breast-fed infants are attacked by diarrhea when the mother or the nurse becomes angry, or suffers other psychic disturbance, or with the occurrence of menstruation. At times also a sort of idiosyncrasy is observed—one child not tolerating the milk of a given nurse, while another child may thrive upon it. **Children of the lower classes** suffer most commonly from acute gastro-intestinal catarrh, because less attention is devoted to their care. The poor also cannot always provide proper milk on account of the expense. It is, therefore, not remarkable that the disease is particularly prevalent in **large cities.** **Illegitimate children** perish in large number, because they are placed at board at the smallest possible cost, and in consequence are poorly nourished and cared for. Such foster-mothers are appropriately designated "angel-makers."

Symptoms and Diagnosis.—The earliest symptoms of acute gastro-intestinal catarrh in infants often consist in frequent **eructation** and **vomiting.** The vomited matter at first contains lumps of casein; but if the vomiting has persisted for some time the milk is returned uncoagulated, obviously because the gastric juice has become too deficient in acid to effect coagulation of the milk. Soon **diarrhea** sets in, and in some cases it may be the earliest symptom; or, if the bowel principally be involved, it may be unattended with vomiting. The stools become thin, exhale a fetid and acid odor, and are either greenish in color or yellowish when evacuated, but when exposed to the air soon acquiring a greenish hue. If the bowels move with great frequency, the stools present a grayish color, and the condition is then designated cholera infantum. The **abdomen** frequently is greatly distended in consequence of excessive development of gas in the intestine. From time to time rumbling and gurgling sounds—**borborygmi**—are audible, indicating increased intestinal peristalsis and rapid propulsion of the fluid intestinal contents. From time to time the little patient cries aloud, distorts the face in pain, and draws the legs upon the abdomen, in consequence of paroxysmal **abdominal pain—tormina.** Frequently escape of fetid gas follows, not rarely associated with fluid intestinal contents. When the diarrhea has existed for some time inflammatory redness of the skin about the anus and the buttocks develops, because soiling and irritation of the parts by fecal matter are unavoidable. The **secretion of urine** almost ceases entirely. The urine frequently contains albumin and tube-casts and considerable indican. In consequence of the copious loss of water in the stools **thirst** is greatly increased, and the mouth feels sticky and dry. The peripheral portions of the body are usually cool, while the covered portions of the trunk are unusually hot, in consequence of febrile elevation of the **internal temperature.** Unfortunately, rapid and dangerous **collapse** often occurs in the course of the disease. The face becomes shrunken and presents a tired and aged appearance. The fontanels also become depressed, and the margins of the cranial bones can be slightly moved upon one another, the voice becomes faint and feeble, movement is painful and difficult, and the action of the heart is greatly accelerated. In consequence of progressive anemia of the brain cerebral manifestations appear that have been designated *hydrocephaloid.* At the same time the little patient becomes more and more apathetic, lying with its eyes half closed, and presenting tonic twitchings of the face or in the extremities; finally, general clonic spasms may occur and death result.

In rare instances the exposed *cornea* becomes desiccated and it may undergo rupture.

Anatomic Alterations.—In the acute gastro-intestinal catarrh of infants the statement already emphasized in connection with acute intestinal catarrh, that the most intense symptoms during life are often associated with only slight alterations in the intestinal mucous membrane, is frequently confirmed. In addition to fluid intestinal contents, not rarely only slight swelling of the lymphatic follicles is found besides, and these are surrounded by a garland of dilated blood-vessels. In other instances the lesions of acute gastro-intestinal catarrh already described are found.

Prognosis.—Acute gastro-intestinal catarrh in infants is a most serious disease, and the prognosis is grave. This depends especially upon the coöperation, the intelligence, and the care, in part upon the means, of those upon whom the nursing of the sick child devolves.

Treatment.—**Prophylactic measures** first of all require thorough discussion and consideration. These apply to the *nourishment of the infant.* The most natural and the best food for an infant is the *breast-milk of its mother or of a wet-nurse.* Unfortunately, however, it is a growing custom that mothers, from motives of convenience or from the unfounded fear that their beauty will suffer in consequence of nursing, do not nurse their children; apart from those instances in which mothers are forbidden by physicians to nurse their children; for instance, at an age below eighteen years, in the presence of profound anemia, pulmonary tuberculosis, inherited nervous disease (epilepsy, neurasthenia, psychopathy), and the like. The employment of a wet-nurse is expensive, and therefore accessible only to families of means. Further, certain precautions should be observed in nourishing infants with the breast-milk of the mother or of a wet-nurse. Infants readily develop gastro-intestinal catarrh when nursing women indulge in acid food, or become greatly excited, or permit coitus, or menstruate. It also happens occasionally that, from causes that elude scrutiny, the infant fails to thrive, although the nurse is apparently faultless, while it bears admirably the milk of another nurse possibly not presenting so good an appearance. Under such circumstances a change should be made in good time. Should acute gastro-intestinal catarrh develop on weaning the infant, there is no other remedy but to restore the child to the breast and to repeat the attempt at weaning at a later period.

In cases in which nourishment at the breast of the mother or of a wet-nurse is not practicable, *feeding with animal milk* is the next best method, and, as a rule, cow's milk is then employed. Goat's milk is less digestible on account of the greater amount of fat and albumin that it contains, and mare's and ass's milk, which more closely resemble human milk in chemic constitution than does cow's milk, are not generally available. The use of cow's milk is attended with two dangers—namely, infection with tuberculosis and undue readiness of decomposition. Tuberculosis is common in cows, and it may readily happen that tubercle-bacilli gain entrance into the milk, especially when tuberculous nodules have developed in the udder, and the milk is thus infective. This danger cannot be averted with certainty, although it can be diminished if the cow whose milk is to be used is most carefully examined for the presence of tuberculosis, and is no longer employed if even suspicious signs are present, and, besides, if the milk is always thoroughly boiled for a considerable length of time before it is used, in order to effect destruction of any tubercle-bacilli or their spores. The suggestion appears worthy of consideration, to use the milk of several cows, in order to attenuate such tuberculous virus as may be present.

In order to prevent, so far as possible, the decomposition of milk, cleanliness of the dairy and of the dairy-utensils should be observed, and cleansing the udder of adherent contaminations before milking, and of the fingers and the hands of the milker, practised. The milk should at once be covered and kept in a cool place. The cows should be supplied with dry fodder. In large cities *establishments for the supply of infants' milk* exist, which engage in the preparation of milk for infants in accordance with the principles outlined. Milk is also sterilized by many establishments, and is sold in this condition. As may be understood, the greater labor expended in obtaining such milk entails a higher price for the product, and therefore the poor are scarcely able to purchase this specially prepared article. In this connection private philanthropy may render a great service by dispensing such milk under proper conditions. To sterilize milk in private dwellings, the use of the apparatus of Soxhlet may be especially recommended. Under all conditions milk for children should be thoroughly boiled for a considerable length of time after its reception, and be introduced into a clean vessel, which is covered with a glass plate and is placed in a cool spot, and if possible preserved upon ice. The addition of a tablespoonful of lime-water to every quart of milk may also be advised in order to prevent fermentation.

It should be borne in mind that cow's milk differs from mother's milk in containing a larger amount of solids, particularly casein; while, on the other hand, it contains less sugar. In order to make the former more closely resemble the latter it is necessary to add water and sugar to cow's milk. Even then the resemblance between the two is not complete, the casein of cow's milk coagulating in large masses, while that of mother's milk coagulates in fine flakes. In consequence, mother's milk is more readily accessible to the action of the gastric juice and is more digestible than cow's milk. By the addition of lime-water to cow's milk its casein will be precipitated in small flakes.

Children in the first months of life should receive cow's milk and water in equal parts. Then, gradually, one-tenth less water is added, until from the fifth month the child will receive pure cow's milk. To each 250 c.c. of milk a pinch of sugar should be added. The use of sugar of milk is not necessary.

For the preservation of the health of the infant it is further important to regulate the *amount of food* and the *time of feeding*. The stomach and the intestines should be protected from overloading. In the first two months of life an infant should receive food every two, and thereafter every three, hours. The last meal should be given at ten o' clock at night and the first at six in the morning. It is further important to keep the feeding-bottles and the nipples clean. The bottle is thoroughly cleansed with water and salt after each nursing, and is kept filled with water until the next meal. Also, the nipples, in which germs readily lodge, are thoroughly rubbed externally and internally with salt, and are kept in water. Care should further be taken to have the mouth of the infant cleansed after each nursing with a bit of soft, clean linen that has been dipped in water, as, otherwise, remnants of milk may readily undergo fermentation in the mouth, and if they be swallowed they may excite fermentative processes in the stomach. It is understood that the milk should be given not cold, but at the temperature of the body, being warmed in the bottle at each nursing by being placed in hot water.

The feeding of an infant with substitutes for milk—as, for instance, Liebig's, Nestle's, Kufeke's, Löfflund's foods, Biedert's cream-mixture, Gärtner's fat-milk—is not worthy of commendation, because children thus nourished frequently become rachitic and also suffer readily from intestinal catarrh. If acute gastro-intestinal catarrh has developed in an infant, it is wise to withhold milk for from one to three days, and to replace it by pure water, to which a little red wine or Cognac has been added. Should the disease be protracted, Nestle's food, beef-tea, or mucilaginous soups of oatmeal-gruel, sago, or arrow-root may be given instead of milk. Among medicaments calomel deserves first place:

℞ Mercurous chlorid,	0.01 (⅙ grain);
Sugar,	0.5 (7½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

Among **styptics**, colombo-root (infusion of colombo-root, 10.0 : 100—2½ drams : 3 fluidounces; 5 c.c.—1 teaspoonful—every two hours), cascarilla-bark (infusion of cascarilla-bark, 10.0 : 100—2½ drams: 3 fluidounces; 5 c.c.—1 teaspoonful—every two hours), and silver nitrate (solution of silver nitrate, 0.1 : 100—1½ grains: 3 fluidounces; 5 c.c.—1 teaspoonful—every two hours) may be mentioned. Preparations of opium should not be prescribed, because children readily develop alarming toxic symptoms from their use. Of late, **disinfectants** (lactic acid, carbolic acid, benzol, naphthalin, thymol, resorcin, etc.) have been much used, but we fail to see any particular advantage in them over the remedies already recommended. If signs of cerebral anemia appear, strong wine in small doses should be administered, and the head should be lowered.

CHRONIC INTESTINAL CATARRH.

Etiology.—Chronic intestinal catarrh not rarely develops in the sequence of acute intestinal catarrh if this be frequently repeated, and especially if recurrence take place after incomplete recovery from the preceding attack. At other times it develops as an **independent affection,** and under such circumstances definite causes will be found to have been operative. *Hypostatic catarrh* exhibits a marked tendency to pursue a chronic course. It may be induced by the general stasis attending chronic disease of the heart or of the respiratory apparatus, or it may develop as a result of stasis in the portal circulation, and most commonly in association with cirrhosis of the liver. **Chronic debilitating disease,** as, for instance, pulmonary tuberculosis, carcinoma, leukemia, protracted suppuration, often maintains chronic intestinal catarrh. Not rarely **chronic intestinal diseases** (carcinoma, hemorrhoids, tuberculous ulceration, parasites) are accompanied by chronic intestinal catarrh. Chronic constipation is often a cause of chronic intestinal catarrh.

Anatomic Alterations.—Intestinal mucous membrane the seat of chronic inflammation is conspicuous for its **color.** It is brownish-red or grayish-red in appearance, especially on the villi and at the summit of its folds. At times the brownish hemoglobin has been transformed into black melanin, so that the mucous membrane presents a mottled appearance—so-called **villous melanosis.** Besides, the presence of **excessive secretion** is conspicuous, the mucous membrane being covered with transparent or slightly turbid mucus. The **lymph-follicles** are often enlarged, and surrounded by a brownish or blackish zone of pigment. As a rule, the mucous membrane is thickened in consequence of inflammatory hyperplasia of the connective tissue, and often the muscularis and the mucosa also participate in the hyperplasia and thickening. Under such circumstances the condition can be designated *hypertrophic chronic gastric catarrh.* In contradistinction from this, *atrophic chronic gastric catarrh* is attended with abnormal attenuation of the intestinal wall, together with atrophy of the glandular layer of the mucous membrane. Further, especial care should be taken in reaching a decision as to the existence of chronic atrophic intestinal catarrh, as frequently the condition of intestinal atrophy is simulated by insignificant postmortem alterations. Among the *complications* of chronic intestinal catarrh *catarrhal ulceration of the bowel* may be mentioned, which may have resulted from disintegration of lymph-follicles—*follicular intestinal ulceration*—or from destruction of the tissue of the mucosa itself—*catarrhal ulceration of the mucous membrane.*

Among rarer occurrences *polypoid hyperplasia of the mucous membrane* and *cystic degeneration of the glands of the intestinal mucous membrane* may be mentioned.

Symptoms and Diagnosis.—**Alterations in the stools** constitute the main symptom of chronic intestinal catarrh. As a rule, there is irregularity in the movement of the bowels, and often constipation and diarrhea alternate with each other. The abundant presence of mucus in the stools is distinctive. In the presence of catarrh of the large intestine the individual fecal masses are often enclosed in a layer of mucus. In the presence of catarrh of the small intestine, on the other hand, there is intimate admixture of fecal matter. Often the stools are stained yellow by biliary coloring-matter, and on addition of nitric acid they yield the reaction of biliary coloring-matter. At times peculiar, swollen and transparent, sago-like granules are present in the stools, which consist in part of swollen carbohydrates and in part of mucus. These were formerly considered erroneously as distinctive of follicular ulceration of the bowel. The stools at times contain a strikingly large amount of undigested food. If this be recognizable with the unaided eye, the condition has been designated *lientery*. Microscopic examination of the stools discloses, in addition to remnants of food and bacteria, isolated round cells and epithelial cells. The latter are often peculiarly swollen, and, in the presence of catarrh of the small intestine, bile-stained.

Rumbling in the abdomen—borborygmi, distention of the abdomen—intestinal meteorism, and **abdominal pain—tormina**—are frequently recurring symptoms, which at times become particularly severe. Chronic intestinal catarrh is usually **unattended with fever. Nutrition** may suffer in marked degree. The patients emaciate progressively, and acquire a grayish and sallow appearance. **Psychic alterations** occur with especial frequency, and they have not improperly been explained as the results of auto-intoxication, inasmuch as abnormal metabolic products are absorbed from the intestine and derange the functions of the nervous system. The patients become hypochondriacal, believe themselves to be suffering from all possible serious diseases, develop a fear that they will become insane, are involved in doubts as to their mental and physical ability, and complain of all possible conditions of fear. They are often troubled by annoying vertigo—**intestinal vertigo,** which can at times be developed voluntarily by pressure upon the bowel. Palpitation of the heart and asthmatic states also occur occasionally. The **appetite** is variable, and is impaired especially when the stomach is also involved in the inflammatory process. **Thirst** is usually increased. The **urine** is usually voided in small amount, is therefore generally characterized by its dark-red color, and frequently precipitates a reddish, granular sediment of urates.

The *duration of chronic intestinal catarrh* is subject to wide variations. In some individuals it persists throughout life. If ulceration of the intestinal mucous membrane has taken place, hemorrhage, rupture, and, after cicatrization of ulcers, stenosis of the bowel may occur as *complications*, but these occurrences are rare. Most commonly chronic inflammation of the bowel involves the ileum and the colon, in correspondence with the like conditions that attend acute intestinal catarrh, and what has been said in the preceding refers principally to chronic catarrhal ileocolitis.

Chronic catarrhal duodenitis is characterized by involvement of the choledoch duct and *chronic catarrhal jaundice*. *Chronic inflammation of the rectum—chronic catarrhal proctitis*—like acute proctitis, is attended with a persistent *desire for stool—anal tenesmus*. The stools at times consist only of mucus or of mucus and pus. Sometimes the structures surrounding the rectum become involved in the inflammatory process—*periproctitis;* and if a collection of pus in this situation ruptures outward or inward, or in both directions simultaneously, an external or an internal or a complete *rectal fistula* will result.

Prominence should be given to that form of colitis which has been designated *membranous enteritis—mucous colic*. This is characterized generally by the periodic evacuation of whitish membranes, tubes, and shreds, consisting of mucus, and less commonly of fibrin and albuminoid material, and which have collected along the bands of the colon. .The evacuations are usually preceded by disagreeable sensations in the abdomen, with distention, and the expulsion of the sometimes long casts is attended with severe pain and difficulty. Care should be taken, further, to avoid confounding such casts with tendinous structures, the fibrous remains of asparagus, and the like, and this can readily be done by microscopic examination. The membranes and tubes exhibit under the microscope a swollen structure, which becomes striated on addition of acetic acid, and contains epithelial cells, isolated round cells, and crystals of triple phosphate and cholesterin. This condition occurs principally in nervous individuals, particularly women, and while not dangerous is often very obstinate. Only in the minority of cases does the condition appear to be the sequel of chronic intestinal catarrh—true membranous enteritis; while in the majority of cases it appears to be a secretory neurosis of the intestinal mucous membrane—mucous colic.

The occurrence in children suffering from chronic intestinal catarrh of stools containing considerable fat has been described as *fatty diarrhea*, the absorption of fat being obviously much diminished. The stools present a yellowish or grayish appearance, give off an odor of fatty acids, when rubbed up with water and examined microscopically exhibit an abundance of fat-globules, and on analysis are found to contain 67 per cent. instead of 20 per cent. of fat. The condition is observed especially in association with catarrh of the small intestine, and also when the pancreas is diseased and the discharge of bile into the intestine is interfered with.

Prognosis.—Although chronic intestinal catarrh is as a rule not a dangerous disorder, it is often extremely obstinate and annoying. At times the prognosis is unfavorable, because the causative conditions (carcinoma, tuberculosis) are incurable.

Treatment.—In the treatment of chronic intestinal catarrh consideration should first be given to the causative conditions, and an effort made to relieve them—**causal therapy. Symptomatic treatment** will have to be directed most frequently to the relief of existing *constipation*. The **mode of life** and the **diet** will next require correction. The patients should take walks regularly, indulge in gymnastic exercises and physical activity, and never suppress the desire for stool, however slight this may be (a rule that must be especially impressed upon women), while a daily effort to evacuate the bowel at the same time should be encouraged, even in the absence of the normal desire, in order to accustom the bowel to habitual activity. The diet should include such articles of food as experience has shown to be capable of stimulating the bowels to increased activity. Among these may be mentioned Graham bread with butter and honey in the morning, Graham bread with butter in the evening, fruit at noon and in the evening, and vegetables at noon. Often the patients have themselves discovered certain **domestic remedies** capable of inducing movement of the bowels, as, for instance, the ingestion of a glass of cold water when the stomach is empty, or the smoking of a cigar. Among medicaments, **laxatives, mineral waters,** and **enemata** may be mentioned. It should be pointed out that the intestine readily becomes accustomed to a given medicament or procedure, so that variation is necessary from time to time. Among laxatives, rhubarb-root, senna-leaves, aloes, jalap, and podophyllin particularly may be recommended; as, for instance:

℞ Aloes,
Compound extract of rhubarb, each, 1.5 (22½ grains);
Powder and fluid extract of licorice, sufficient to make 30 pills.
Dose: 1 or 2 pills at night.

℞ Aloes,
Compound extract of rhubarb,
Powdered jalap, each, 1.0 (15 grains);
Powder and fluid extract of licorice, sufficient to make 30 pills.
Dose: From 1 to 3 pills at night.

℞ Aloes,
Compound extract of rhubarb, each, 1.5 (22½ grains);
Podophyllin, 0.3 (4½ ");
Powder and fluid extract of licorice, sufficient to make 30 pills.
Dose: 1 or 2 pills at night.

℞ Compound licorice-powder, 100.0 (3 ounces).
Dose: 1 or 2 teaspoonfuls in a wine-glassful of tepid water in the morning.

℞ Electuary of senna, 50.0 (1½ ounces).
Dose: 1 teaspoonful in the morning.

Among *mineral waters* may be mentioned the various *bitter waters* (Ofen, Pullna, Saidschütz, Friedrichshall, Birmenstorf, Baden), of which one or two wine-glassfuls should be taken in the morning when the stomach is empty, to be followed in a short time by an equal quantity of cold water. Carlsbad salt (solution of artificial Carlsbad salt—1 or 2 teaspoonfuls to a glass of water when the stomach is empty) is often employed with success. *Enemata* may be administered advantageously in the form of *intestinal infusions.* For this purpose either an irrigator with a tube and a rectal nozzle, or the funnel-apparatus of Hegar, consisting of a large glass funnel with a rubber tube and rectal nozzle, is employed. The irrigating fluid should be water at room-temperature, and from 1 to 2 liters should be permitted to flow into the large intestine. The introduction is best effected with the pelvis elevated, and even more satisfactorily in the knee-elbow posture. Frequently the water will not flow because the rubber tube is filled with air. This can be removed readily by repeated and vigorous pressure with the fingers upon the tube. Russian physicians were the first to recommend large *enemata of oil* for the relief of obstinate constipation (from 400 to 500 c.c. of poppy-seed oil—about 1 pint—to each enema). Glycerin-enemata are also highly efficacious (2.0—30 minims—glycerin to each enema), while glycerin-suppositories are less reliable in action. Well-to-do patients may during the summer undertake courses of treatment at the *springs.* Among these, the springs of Carlsbad, Marienbad, Tarasp, Kissingen, Homburg, Ems, Wiesbaden, and Vichy especially should be mentioned. In cases of *chronic duodenitis with jaundice* all articles of food containing fat should be interdicted. *Membranous enteritis* is often resistant to all forms of treatment. In the first place, the nervous general condition should be taken into consideration, and in addition intestinal infusions of oil, water, and astringents should be employed.

INFLAMMATION OF THE CECUM AND THE VERMIFORM APPENDIX AND THE SURROUNDING TISSUES (TYPHLITIS; APPENDICITIS; PERITYPHLITIS; PARATYPHLITIS).

Etiology and Anatomic Alterations.—Inflammatory processes not rarely occur in the right iliac fossa, which have certain important symptoms in common (usually **sudden onset, pain,** and **acute tumor-formation**). Inflammation of the cecum is known as *typhlitis,* while inflammation of the vermiform process is designated *appendicitis.* Circumscribed inflammation of the peritoneal covering of the cecum or the vermiform process is known as *perityphlitis.* Inflammation of the retrocecal connective tissue, by which the vermiform appendix is attached to the iliac fossa, is known as *paratyphlitis.* Typhlitis and appendicitis are almost always independent diseases, while perityphlitis and paratyphlitis are usually of secondary origin, and most commonly result from preceding appendicitis or typhlitis. *Appendicitis* and *perityphlitis* are the conditions most commonly encountered. In the majority of cases the process pursues the following course: Fecal matter becomes lodged in the vermiform appendix, and in consequence of desiccation becomes solidified into so-called *false fecal concretions,* which after a while cause mechanical irritation of the walls of the appendix, with secondary inflammation and necrosis. The inflammatory process readily extends to the adjacent peritoneum, so that to appendicitis is superadded perityphlitis. The latter frequently occurs from perforation of the walls of the appendix by a fecal concretion, which may become lodged in the orifice of perforation or is expelled into the peritoneal cavity. At times the free extremity of the vermiform process is detached in its circumference, the stone occupying the mouth of the appendix. Perityphlitis usually gives rise to the formation of an encapsulated, purulent exudate, which may acquire a fecal odor even if perforation of the bowel has not taken place. After perityphlitis has existed for a considerable length of time adhesions are usually so numerous that it is difficult to find the appendix at all, not to speak of the point of perforation and a fecal concretion.

Clinical experience has shown that the tendency to the formation of fecal calculi, and therefore also to appendicitis and perityphlitis, is unevenly distributed. In some families the disease occurs with such remarkable frequency that the causative factors have been looked for in some **anatomic peculiarity of the vermiform appendix** (excessive length, undue development of Gerlach's valve at the entrance to the vermiform appendix, unusual size). Without doubt the character of the **food** has an influence upon the formation of fecal concretions, as these are observed most commonly in individuals who live excessively upon a vegetable diet. The swallowing of **foreign bodies** is also a dangerous practice in this connection. Thus, I have observed fecal concretions formed about swallowed hair from the beard and bristles from the tooth-brush. A **sedentary mode of life** predisposes in marked degree to the formation of fecal concretions and their consequences; so does also **constipation.**

As several of the causative factors named are operative in the individuals concerned throughout life, it can be readily understood that appendicitis and perityphlitis are frequently recurrent. Attention has recently been called to the fact that inflammatory conditions (catarrhal, hemorrhagic, purulent, gangrenous) of the **mucous membrane of the vermiform process** are not uncommon, and that these also may be followed by the formation of fecal concretions, appendicitis, and perityphlitis. **True fecal concretions** constitute much less commonly than false fecal concretions the starting-point for inflammation of the vermiform appendix and its vicinity. True fecal concretions consist of mineral deposits, whose principal constituents are generally calcium phosphate, calcium carbonate, and ammonio-magnesium phosphate. At times **swallowed foreign bodies** give rise to appendicitis and perityphlitis. Thus, a railway-official under my care died with pyemic manifestations in the sequence of appendicitis and perityphlitis, who attributed his disease to the sharp fragment of a tooth-pick that he had swallowed. It should not be forgotten that at times **ulceration of the mucous membrane of the vermiform appendix** (catarrhal, typhoid, tuberculous, carcinomatous, actinomycotic) may be followed by appendicitis. Not rarely *traumatism*, *exposure to cold*, and, in my experience, with especial frequency, the *ingestion of cold fluids* (beer) are named as causes of the disease, but these influences probably play the *rôle* rather of **contributory factors**, which furnish the final impulse for the outbreak of the disorder, long after the true germ had been present.

Although appendicitis is the most common cause for perityphlitis, it is by no means the sole cause. Perityphlitis may, for instance, be dependent upon a preceding **typhlitis.** At times it originates from a **perimetritis, a salpingitis,** or an **oophoritis,** and it may happen that the exudate has already disappeared from the actual starting-point of the inflammation, and an apparently primary perityphlitis is encountered. In women it is, therefore, not uncommon for the manifestations of perityphlitis to appear in the sequence of menstrual disturbances. *Constipation* was formerly considered the cause of *inflammation of the cecum* (*typhlitis*), and the condition was, accordingly designated *stercoral typhlitis.* Stagnation of fecal matter may readily take place in the cecum, because here the intestinal contents become inspissated, and to a certain degree must be propelled against the force of gravity. A sedentary mode of life and excessive use of vegetables, as well as swallowed foreign bodies, will favor fecal stagnation. Recently attempts have been made to cast doubt upon the occurrence of stercoral typhlitis, but upon the basis of personal experience it appears to me that they have not succeeded. *Ulcerative processes* may induce inflammation in the cecum in the same manner as in the vermiform process. **Traumatism** is at times followed by typhlitis.

Paratyphlitis may arise by extension from typhlitis or perityplitis, or it may develop in the sequence of paranephritis, parametritis, periproctitis, psoas-abscess, inflammation of the innominate bone or of the vertebral column, when the inflammatory process extends to the iliac fossa or gives rise to burrowing abscesses therein. As in perityplitis, so also in paratyphlitis it may happen that the exudate at the site of primary inflammation has been absorbed, leaving a paratyphlitis. At times paratyphlitis develops in the sequence of **infectious diseases** (typhoid fever, septicemia). *Traumatic paratyphlitis* may occur as an independent disorder, as after the lifting of heavy weights, after over-exertion in the process of washing and the hanging up of linen, and in some persons it may be recurrent. The occurrence *of refrigeratory* (*rheumatic*) *paratyphlitis* may properly be doubted.

The inflammatory processes under consideration may develop at any period of life, though most commonly *between the sixteenth and the twenty-fifth year,* and the impression has been created that their frequency has become progressively greater within recent times. Men are attacked, experience has shown, more than twice as often as women.

Symptoms.—*Appendicitis* and *perityphlitis* frequently set in with sudden, sharp **pain in the right iliac fossa,** the intensity of which is often so great that the patients will not tolerate the slightest pressure with the fingers, not even that of a light sheet. The *abdomen* is generally distended, obviously because a large amount of gas has accumulated in the small intestine. Often a **considerable prominence** is noticeable in the **right iliac fossa.** On palpation, which naturally should be practised with great care, only increased tension of the abdominal walls and increased resistance without sharp limitation, may at first be appreciated. In the course of a few days at most, a readily circumscribed *tumor* will have formed in the *right iliac fossa.* The most common variety of tumor begins below at about the middle of Poupart's ligament, forming a convex arch toward the median line of the abdomen and disappearing on the right side of the abdomen below the lower margin of the liver. The right lumbar region also is frequently distinctly resistant. Not rarely the tumor is smaller in extent. Palpable exudates of band-like shape also occur.

In addition to the local abdominal alterations there are usually disturbances of gastric and intestinal activity. **Vomiting** is an exceedingly frequent manifestation. The vomited matters consist at first of gastric contents, but after emesis has persisted for some time they assume a bilious character, and present at times a yellowish, and at other times a greenish color. **Eructation—singultus**—is one of the common symptoms. **Appetite** is entirely wanting, while **thirst** is greatly increased. The **tongue** is almost always coated. Often the **breath** is extremely offensive. The **bowels** are usually constipated, although at the beginning of the attack a few thin bowel-movements may be passed. The urine constantly contains an increased amount of indican in consequence of derangement of digestive activity in the small intestine and increased proteid decomposition in the bowel. The urine is at the same time passed in small amount, and consequently is generally dark red in color. Appendicitis and perityphlitis are almost always attended with **fever**; a perfectly afebrile course is exceptional. The elevation of temperature may reach 39° C. (102.2° F.) or 40° C. (104° F.). The fever pursues no definite type, and declines gradually after a variable period of time. The patients usually occupy a peculiarly rigid **posture**, lying upon the back, as a rule somewhat inclined toward the right, and as much as possible avoiding all change in position on account of the resulting severe abdominal pain.

The *duration of an attack of appendicitis and perityphlitis* is extremely variable, and fluctuates between days, weeks, and months. In the natural course of the disease the fever disappears slowly, the pains become less severe, the exudate becomes smaller, harder, and more sharply circumscribed. Painless residues of exudate and infiltrations in the right iliac fossa often persist for many months. Frequently it is remarkable how after a copious evacuation of the bowels the exudate becomes almost suddenly smaller, obviously because the accumulated fecal matter has artificially increased the extent of the exudate. Often sharp pain and tenderness in the right iliac fossa persist for a long time.

Dropsy of the vermiform process develops at times as a sequel of appendicitis, and it may be represented by a smooth and movable tumor as big as a fist. A similar condition may develop when ulcers, and subsequently cicatrices, form in the neighborhood of the entrance to the vermiform appendix, and these give rise to occlusion of the appendix. Mucoid masses then accumulate in the appendix with increasing distention.

Among the *complications*, one of the most dangerous is *diffuse acute peritonitis*, which may be readily recognized from the extensive tenderness of the abdominal walls, the increased distention of the abdomen, and the aggravation of the general condition. At times incautious bodily movement or dietetic error may be found to be a causative factor. Improper manipulation of the abdominal walls also may give rise to diffuse peritonitis, an encapsulated peritiphlytic exudate rupturing into the abdominal cavity. Constipation and expulsive efforts at stool may likewise give rise to general peritonitis. *Pneumoperitonitis* develops when in addition to fecal matter air also enters the abdominal cavity through a perforation in the vermiform appendix. The principal distinctive features of this condition consist in marked abdominal distention, abdominal tenderness, absence of hepatic and splenic percussion-dulness, and general collapse. At times symptoms of *ileus* appear (fecal vomiting, constipation, and absence of flatus) without a mechanical obstruction of the intestine being demonstrable at autopsy. Under such circumstances the condition is probably one of dynamic or paralytic ileus resulting from partial paralysis of the muscular coat of the bowel. Some patients complain of *dysuria*, which is probably dependent upon perityphlitis, and it may become necessary to remove the urine with the aid of a catheter.

I have observed the *urine* to be *alkaline* for days with particular frequency when absorption of the exudate has set in. *Pleurisy* develops in about one-third of the cases. Most commonly it is right-sided, although it may be left-sided or bilateral. Usually it is serous in character. Often it is absorbed with marked rapidity. Among the complications *rupture of the pus* may yet be mentioned. This may take place into the intestine, and its occurrence can be recognized from the presence of pus in the stools. I have on several occasions also observed a desire for stool and pain during evacuation of the bowels—tenesmus ani. In other instances the pus ruptures into the urinary passages (pelvis of the kidney, ureter, bladder), and pyuria and dysuria develop. Rupture into the uterus or the vagina is also conceivable. At times the pus burrows beneath the cutaneous integument. Redness and doughy induration of the skin, increasing prominence and fluctuation of progressively increasing distinctness are indicative of the advent of this condition. The point of rupture is at times in the neighborhood of the right iliac fossa itself, but at other times in consequence of burrowing it may be situated at a remote point, as, for instance, at the umbilicus, below Poupart's ligament, etc.

Occasionally *subphrenic pyothorax*, or *pyopneumothorax*—that is, an accumulation of pus or of pus and air between the diaphragm and the upper surface of the liver—has been observed to develop in the sequence of perityphlitis. At times appendicitis and perityphlitis give rise to *septicopyemia*, purulent thrombi forming in the mesenteric veins, from which emboli may be detached and be swept into the circulation, giving rise especially to multiple abscess of the liver. In one case I observed such a condition develop seventeen years after an attack of acute appendicitis.

Stercoral typhlitis usually occurs after preëxisting constipation. The patient complains suddenly of pain in the right iliac fossa, and in this situation is found a painful tumor, which, in contradistinction from a perityphlitic exudate, yields a dull, and not a dull-tympanitic note upon percussion, and does not extend to the median line of the abdomen and upward to the lower margin of the liver, but which at times, when marked pressure is made with the finger, presents depressions due to indentations of the fecal matter. It is further distinctive that after a copious evacuation of the bowels the tumor usually disappears, and at most slight induration remains for a short time, which may possibly be dependent upon inflammatory swelling of the intestinal mucous membrane. The abdomen is intensely distended, in consequence of fecal stagnation and abundant generation of gas. There is no increase in the amount of indican in the urine, or this is but slight. Febrile movement also is wanting, or is not marked. As a rule, a rapid change for the better occurs when a copious bowel-movement has been induced; otherwise, naturally, serious dangers may arise, among which may be mentioned *ileus*, *perityphlitis*, *peritonitis*, and *rupture of the bowel.*

Paratyphlitis also is attended with *pain* and *tumor-formation in the right iliac fossa*, but the tumor is not so superficial as in the case of perityphlitis and typhlitis, and it is concealed by the tympanitic percussion-note yielded by the cecum. The amount of indican in the urine is not increased. Fever is present, and the patient usually keeps the *right thigh* flexed, probably in consequence of involvement of the iliopsoas. At times unyielding muscular contractures develop. Occasionally pressure exerted by the exudate upon the nerves to the extremity passing through the pelvis gives rise to *paresthesia*, *neuralgia*, and *paralytic conditions in the right leg.* Pain in the right testicle and spasmodic elevation of this organ have been observed. Pressure of the exudate upon the veins of the extremities may give rise to *edema* in these parts. The disease is not unattended with *dangers* and other *complications.* Thus, general *septicopyemia* may develop, or the *pus may rupture* into the intestine, the urinary passages, the peritoneal cavity, or externally. In the last-named event extensive burrowing of the pus may take place, as, for example, down into the extremities, the perineum, the vagina. In one case I observed rupture of the pus upon the left side of the abdomen. It may also occur that the pus in the right iliac fossa has been absorbed, and that suppuration is present only at the most dependent point. In this way paratyphlitis may be transformed into paranephritis, parametritis, or periproctitis. Rupture of the pus into the peritoneal cavity is particularly dangerous, as it is usually followed by fatal peritonitis. After rupture of the pus into the bladder or the bowel recovery not rarely takes place, because the fistula is so constituted that while it permits the escape of pus it prevents the access of urine or feces to the inflammatory focus, with the development in it of putrefaction. At times *perityphlitis* is superadded to paratyphlitis without preceding rupture of the pus. Paratyphlitis frequently pursues a chronic *course*, and the patient at times dies from progressive exhaustion.

Diagnosis.—With regard to the differentiation between appendicitis, perityphlitis, paratyphlitis, and typhlitis the essential facts have already been stated. Attention should, further, be directed to the circumstance that these diseases may at times be confounded with other disorders. **Carcinoma of the cecum** usually occurs in old persons, gives rise to the development of a nodular tumor, induces rapid marasmus, begins insidiously, and pursues a chronic and often afebrile course. **Invagination of the bowel** also gives rise to a palpable tumor and to constipation, although the latter is usually preceded by thin bloody and mucous stools. **Fecal impaction—coprostasis**—is unattended with inflammatory manifestations (pain, fever). **Tuberculosis of the vertebral column** or of the **pelvic bones** is at times attended with accumulation of pus in the right iliac fossa, but each is usually associated with alterations in the vertebral column or the pelvic bones respectively. **Psoitis** is unattended with disturbance in intestinal activity. **Carcinoma** and **tuberculosis of the mesenteric glands** give rise to the formation of multinodular tumors. At times it is impossible to differentiate appendicitis from **salpingitis**. Confusion with **biliary** or **renal colic** can be avoided if jaundice or alterations in the urine be present. *Wandering kidney* constitutes a smooth, bean-shaped, usually movable tumor, without tenderness on pressure.

Prognosis.—The prognosis of the inflammatory disorders under consideration is not unfavorable. It is the more favorable the more certain the diagnosis, and the more clearly the clinician constantly has in mind the therapeutic measures to be employed.

Treatment.—*Appendicitis* and *perityphlitis* should be treated, like acute peritonitis, with **rest in the recumbent posture, a liquid diet** (milk, beef-broth with egg, coffee with milk, tea with milk, dilute wine), and **opium**:

℞ Powdered opium,	0.03 (½ grain);
Sugar,	0.3 (4½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

An **ice-bag** that is not too heavy should be applied over the right iliac fossa. When the acute inflammatory manifestations have subsided and the exudate has been well circumscribed, the ice-bag should be replaced by a **hot cataplasm**, which will better favor the absorption of the pus. Severe pain will frequently be relieved speedily by the application of from 6 to 10 **leeches**. Treatment with opium should be persisted in until all pain in the right iliac fossa has disappeared, even if many weeks should be required. Some persons, however, complain of nausea and increased pain after the administration of opium. Under such circumstances the opium should be replaced by **subcutaneous injections of morphin**:

℞ Morphin hydrochlorate,	0.3 (4½ grains);
Glycerin,	
Distilled water,	each, 5.0 (75 minims).—M.

Dose: 0.5 (8 minims) subcutaneously twice daily.

Painless residua of exudate may be made to disappear by means of daily **saline baths** (5000 grams of salt to a full bath at a temperature of 28° R.—95° F.), and by covering the skin in the right iliac fossa with a flannel cloth smeared with **mercurial ointment** or with **potassium-iodid ointment**, or with an ointment of **iodoform** or of **ichthyol**. Massage may also be practised cautiously. Especial consideration should be given to the question as to whether, and how often, existing *constipation* should be relieved. In the presence of acute inflammatory manifestations the constipation should be permitted to continue, but also subsequently it will be sufficient if the bowels are moved once every eight days. This end can be best attained by means of a **glycerin-enema** (2.0—30 minims—glycerin to the enema), whose action remains rather local. To avoid *recurrences* of the disease certain **dietetic regulations** must be observed (avoidance of an excess of vegetable food, daily exercise in the open air, care to secure a daily evacuation of the bowels, and the like). At times appendicitis and paratyphlitis will require **surgical intervention**, and this indication may arise either when an abscess has formed *in the peritoneal cavity*, or if rupture of pus into the abdominal cavity has taken place, or, finally, in cases of *recurrence* of the disease. The contention of some surgeons that appendicitis and perityphlitis are always surgical diseases is an error, as most cases pursue a favorable course with internal treatment.[1]

Incision of an encapsulated abscess in the peritoneal cavity becomes necessary in order to avoid rupture of the pus, although it is often difficult to elicit deep fluctuation with certainty through the tense abdominal wall. Although exploratory puncture is considered by some physicians as wholly free from danger, we can by no means coincide with this view, and fear not without reason the possibility of accidental puncture of the intestine, and fecal and putrid infection of the inflammatory products. The abdominal cavity has also been opened after *rupture of the pus into the peritoneal cavity*, and *extensive perforative peritonitis*, the fecal and purulent masses removed by sponging (toilet of the peritoneum), and when possible also the appendix excised. Repeatedly recurring perityphlitis has been treated by *resection or extirpation of the vermiform appendix*. Most surgeons select for the performance of the operation a period when the inflammatory exacerbation has come to an end, while others operate at the time of the attack. In general, the first method mentioned is to be preferred, and the latter should be selected only when the attack itself demands surgical intervention.

Stercoral typhlitis requires the administration of **purgatives**. *Intestinal infusions of cold water* by means of Hegar's funnel-apparatus may be recommended, because their action is rather restricted to the large intestine. It may happen that the first infusions will be unattended with the evacuation of any fecal matter, so that the water returns from the bowel almost unchanged. Under such conditions it is advisable to repeat the infusion every three hours, in order to soften the excessively inspissated fecal matters, and to facilitate their progress in the intestine. Even if copious evacuation of the bowels have taken place, the infusion should be repeated upon the following days in order to empty the bowel as thoroughly as possible. In addition, a **hot cataplasm** should be applied over the right iliac fossa for the control of the inflammatory manifestations. The patient must thereafter secure regular evacuation of the bowels.

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[1] With these views the consensus of clinical opinion is scarcely in accord. The results from the medicinal treatment of appendicitis are at least uncertain. The administration of an opiate is likely to mask the symptoms and induce a false sense of security. Should the bowels remain constipated despite the administration of a laxative, and especially if vomiting be present, operation should be resorted to.—A. A. E.

Paratyphlitis is rather a surgical disease. An effort should be made by means of **hot cataplasms** to induce absorption of the inflammatory products, but should fluctuation appear **incision of the accumulation of pus** should be undertaken.

ROUND ULCER OF THE DUODENUM.

Round ulcer of the duodenum is a rare disorder whose development, manifestations, and treatment coincide with those of round ulcer of the stomach. The condition is attended with a *sharply circumscribed loss of tissue* in the mucous membrane of the duodenum of circular outline, and resulting from the digestive activity of gastric juice that has entered the duodenum whenever local stagnation has taken place in the blood-vessels of the mucous membrane. The lesion may thus be designated a *digestive* or a *peptic ulcer*. Duodenal ulcers are almost unexceptionally found above the point of entrance of the choledoch duct into the duodenum, most frequently in the upper horizontal segment, for below the entrance of the choledoch duct the gastric juice becomes inactive, because the bile causes precipitation of the pepsin, and the alkaline intestinal and pancreatic juices neutralize the hydrochloric acid of the gastric juice. Nothing definite is known with regard to the *causes* of round ulcer of the duodenum. In numerous instances the condition has been observed to develop after *burns of the skin*. Experience has shown that, in contrast with round ulcer of the stomach, duodenal ulceration occurs *more commonly in men* than in women. *Pain* in the right upper quadrant of the abdomen is quite a constant symptom, and in the differentiation from round ulcer of the stomach emphasis has been placed upon the fact that this pain does not succeed immediately upon the ingestion of food, but it appears only after the lapse of from two to four hours, when the gastric contents enter the duodenum. Often *duodenal hemorrhage* takes place. This most frequently gives rise to *bloody stools*, and much less commonly also to *hematemesis*. *Perforation of the ulcer* is a serious danger, and this may take place either free into the abdominal cavity, and then give rise to rapidly fatal *perforative peritonitis*, or into a previously encapsulated cavity. Two of my patients died at an advanced age of *carcinoma of the duodenum*, which developed at the margins of a round ulcer of the duodenum. In one of the cases metastatic growths developed in the spinal cord, with pressure-paralysis, and in the other secondary carcinoma of the liver occurred. Should cicatrization of a duodenal ulcer take place *cicatricial stenosis* *of the duodenum,* and above the constriction *dilatation of the duodenum, of the stomach, and even of the esophagus,* may result.

The **prognosis** is grave under all conditions. At times ulcer of the duodenum develops insidiously, and causes death within a short time from uncontrollable hemorrhage or from perforative peritonitis.

The **treatment** is the same as that for round ulcer of the stomach (pp. 220–222).

CARCINOMA OF THE INTESTINE.

Etiology and Anatomic Alterations.—Nothing definite is known with regard to the *causes* of carcinoma of the intestine, and the knowledge must suffice that carcinoma of this organ, like that of other organs, is a *disease of advanced years.* Only rarely does the disorder appear before the fortieth year of life. That in a small number of cases it may develop from an **ulcer of the intestine** has already been pointed out in the preceding section. Carcinoma of the intestine occurs more commonly in *men* than in women.

Almost always the *new-growth is primary.* Secondary carcinoma of the intestine is rare. Most commonly the new-growth is seated in the **large intestine,** and particularly in the rectum. The **flexures of the bowel** are attacked with especial preference, perhaps because in these situations the mucous membrane is subjected to particularly active mechanical irritation by the advancing column of fecal matter. Accordingly, the sigmoid flexure, the hepatic flexure, and the splenic flexure of the colon and the cecum are the principal seats of carcinoma of the bowel. The most common form of carcinoma of the intestine is the *annular variety,* the entire circumference of the bowel being involved in the new-growth. Accordingly as the carcinomatous tissue is dense and deficient in fluid, of medullary consistency, or traversed by cavities with gelatinous contents, a distinction is made, as with carcinoma in other organs, between *scirrhous, medullary,* and *alveolar carcinoma.* Histologically the new-growth is a *cylindrical-cell carcinoma,* as its cellular elements result from abnormal proliferation of the epithelial cells of the glands of the intestinal mucous membrane. Disintegration of carcinomatous tissue gives rise to *carcinomatous ulcers.* Under such conditions the destruction of tissue may be so extensive that possibly remains of carcinomatous tissue are found only at the margins of the ulcer. This process is attended with two dangers, namely, *hemorrhage from* and *perforation of the intestine.* Should the ulcers undergo cicatrization, stenosis of the bowel results at times in consequence of contraction of the cicatricial tissue. *Dilatation of the intestine* occurs not rarely above the position of the new-growth, because the lumen of the bowel is diminished by the carcinoma or the cicatrix. If, naturally, the carcinomatous tissue has undergone extensive disintegration, the lumen of the bowel may be converted into a large cavity at the site of the new-growth.

Symptoms and Diagnosis.—The diagnosis of carcinoma of the intestine can scarcely be made with certainty unless it has been possible to demonstrate the presence of a **nodular tumor of the bowel,** which has undergone gradually progressive increase in size in a person of advanced years, and is attended with progressive asthenia and anemia. The tumor may be felt either through the abdominal wall or after introduction of the anointed finger into the vagina or the rectum. Carcinoma of the rectum may at times be accessible also to ocular inspection after the *introduction of a rectal speculum.* In addition there are also **changes in intestinal activity.** The patient usually suffers from *constipation* as long as the new-growth diminishes the lumen of the bowel, and as a result the *abdomen may be tensely distended* in consequence of stagnation of fecal matter and gas. At times the *feces present a peculiar shape,* appearing as small, abbreviated columns, like the fecal matter of the sheep or the goat. It is true that the feces acquire a similar appearance also in cases of inanition and in connection with obstinate constipation, if the latter be due to spasmodic contraction of the musculature of the intestine. Should disintegration of the carcinomatous tissue take place subsequently, *diarrhea* frequently occurs. The thin intestinal evacuations not rarely are noteworthy for their putrid and fetid odor, and they contain blood and pus. The discovery in them of fragments of carcinomatous tissue that can be definitely recognized as such on microscopic examination is an exceedingly uncommon event. The patient often suffers from severe **pain** at the site of disease, and this frequently is especially severe at night; the tumor is further exceedingly tender to touch. The urine is generally passed in diminished amount, and it contains an **increased amount of indican.** The **inguinal lymphatic glands** are frequently enlarged and indurated. Although the **appetite** is impaired, **thirst** is generally increased. The **bodily temperature** is frequently elevated if the carcinomatous tissue is in process of disintegration and gangrene, and under such conditions **septic symptoms**—chills—are also observed.

The *diagnosis of neoplasm of the intestine* must not be considered as by any means easy. It is important, in the first place, to localize the new-growth correctly, in order not to confound it with neoplasms of the stomach, the liver, the kidneys, the omentum, the mesentery, and the abdominal lymphatic glands, and with encapsulated peritoneal exudates—perityphlitis. In the differential diagnosis the situation and the mobility of the tumor, and the existence of functional disturbances, should particularly be given consideration. It may be of diagnostic importance to fill the intestine alternately with air and water through the rectum, in order to determine the situation of the tumor and the variations in the percussion-note over it. If a neoplasm has been recognized as arising from the intestine, it next becomes necessary to decide whether it be **carcinomatous** or not. Insidiousness of development, advanced age, and progressive marasmus are suggestive of carcinoma. Especial consideration in the differential diagnosis should be given to **fecal tumors,** which likewise give rise to nodular intestinal swellings. These, however, are unattended with progressive marasmus, and they not rarely permit the production of depressions with the finger, and, above all, they disappear when purgatives are employed for a sufficiently long period. It should, however, be made a rule in the diagnosis of intestinal tumors to pursue a course of purgative treatment, even when the conditions appear clear, as the danger of error in diagnosis is considerable.

The *duration of carcinoma of the intestine* but rarely extends over more than a year, and many patients die in consequence of *progressive asthenia,* after marantic edema or venous thrombi have developed in the lower extremities, or amid *septic manifestations.* At times, in consequence of auto-infection, a comatose state develops rather suddenly, in which the patient dies—*carcinomatous auto-intoxication* or *coma.* At times, however, death results from *complications,* among which *hemorrhage from the bowel, obstruction of the bowel,* and *rupture of the bowel* particularly may be mentioned. As may be readily understood, *hemorrhage from the bowel* may accelerate the loss of strength, especially if it be repeated frequently, and, if it be uncontrollable, death will result from hemorrhage. At times the symptoms of obstruction of the bowel or *ileus* appear suddenly, with insurmountable constipation, absence of flatus, and fecal vomiting. This danger is especially to be feared when the patient is indifferent with regard to regularity in the movement of the bowels or incautiously indulges in indigestible articles of food—leguminous plants or fruit with kernels. *Rupture of the bowel* may take place in various directions, as, for instance, into the peritoneal cavity, when rapidly fatal peritonitis usually follows; into adjacent loops of intestine, when a bimucous intestinal fistula forms; in cases of carcinoma of the cecum into retrocecal connective tissue, with gangrene thereof; into the stomach, the urinary passages, the vagina; or externally, through the skin, with the formation of a *fecal fistula* or *preternatural anus.* At times complications result from the situation of the new-growth in the intestine. Carcinoma of the duodenum not rarely develops at the papilla of Vater, and by pressure upon the choledoch duct, which enters at this point, gives rise to *chronic jaundice.* If the tumor be situated, however, in the upper portion of the duodenum, it will give rise to *dilatation of the stomach.*

Frequently *carcinoma of the rectum* is recognized either not at all or so late that the proper time for operative intervention is permitted to escape. Pain in the sacrum, often radiating to the pelvis and the thighs, mucopurulent and bloody diarrhea, and the development of hemorrhoids in elderly persons should always demand digital examination of the rectum. Often paralysis of the sphincter ani muscle develops, so that putrid fecal matter constantly escapes from the anus, and the patients must protect themselves against the incontinence as best they can by means of linen cloths or paper. Carcinoma of the rectum sometimes pursues a most protracted course. One of my patients died only after the lapse of five years.

It should further be mentioned that *carcinoma of the intestine* may be *latent,* not being recognized at all during life, or under favorable conditions only being suspected to the point of probability from the presence of certain symptoms. Thus, in elderly persons *intestinal hemorrhage* or *ileus* is frequently associated with latent carcinoma of the bowel, as is also *fecal fistula.* At times carcinoma of the bowel is concealed behind a *carcinomatous peritonitis.*

Prognosis.—The prognosis of intestinal carcinoma is unfavorable, for even if an operation will prolong the life of the patient, recurrence or metastasis occurs as a rule, and life cannot be prolonged for any considerable length of time.

Treatment.—Carcinoma of the intestine can be cured only by operative measures through *resection* of the portion of intestine involved in the malignant disease. Naturally, certain limitations surround the employment of the knife. The area involved should especially not be too extensive in length, nor should metastases in other viscera be present. Further, extensive adhesions may render operation not only exceedingly difficult, but even impossible. If resection of the carcinomatous bowel be impossible, the establishment of a *preternatural anus* above the new-growth, or *entero-anastomosis,* is yet available, but this will be indicated only when symptoms of stricture of the bowel appear. In cases of inoperable carcinoma of the rectum *curetting* and destruction of the new-growth with the *galvanocautery* have been undertaken. Internal treatment will be confined to lessening the sufferings of the patient symptomatically and maintaining his strength as fully as possible by means of suitable diet (milk, meat-broth, eggs, wine).

Among the remaining tumors of the intestine *sarcomata* and *polypi* are yet of clinical interest. *Sarcomata of the intestine* give rise to the same symptoms as carcinomata, so that they are indistinguishable from the latter during life. They are much less common than carcinomata, and usually develop as primary neoplasms. *Polypi of the intestine* develop not rarely in children, and are often situated in the rectum. The patients suffer frequently from chronic mucous or *mucous and bloody diarrhea.* Solid masses of fecal matter are sometimes conspicuous from the presence of a *depression* resulting from the pressure exerted by a polyp. Deep-seated polypi may at times protrude from the anus. Sometimes polypi are detached by the pressure of the column of fecal matter and are found in the stools. If the anointed finger be introduced into the rectum, the soft, smooth tumors can often be felt. Removal can be effected only by operative measures. Polypi at a higher level in the bowel at times give rise to *obstruction*, or *intussusception*.

INVAGINATION OR INTUSSUSCEPTION OF THE BOWEL.

Anatomic Alterations.—Invagination of the bowel results from the inversion of one portion of the bowel into an adjacent portion, like the finger of a glove. The invaginated portion is known as the *intussusceptum*, while the external ensheathing portion of bowel is designated the *sheath* or *intussuscipiens*. A distinction is made between the *inner* or *entering*, and the *outer* or *departing, portion* of intestine. The point of reflection from the sheath to the intussusceptum is known as the *upper angle of reflection*, while the point of reflection between the outer and the inner portion of intestine of the intussusceptum is known as the *lower angle of reflection*. A distinction is further made between *descending* and *ascending invagination of the bowel*, accordingly as an upper portion becomes inverted into a lower, or a lower into an upper. All varieties of intestinal invagination do not have clinical significance, and a clear distinction must be made between *vital* and *agonal intussusception*. The latter is unattended with interest, and occurs with especial frequency in children that have passed through a protracted death-struggle or have suffered from diarrhea.

Agonal invagination of the bowel may be recognized from the fact that it is frequently multiple and situated at different parts of the bowel, is often ascending, almost always involves the small intestine, is free from all inflammatory alterations, and can therefore be reduced without difficulty by traction on the bowel.

Vital invagination of the bowel is almost unexceptionally *descending* and *single*. Naturally, together with the bowel the related mesentery is also invaginated, in consequence of which traction is usually exerted upon the invaginated intestine, so that the intussusceptum presents a concavity on its mesenteric aspect and a fissure-like constriction at its lower extremity. It can be readily understood that invagination of the intestine is attended with the danger of *stricture* or *obstruction of the bowel*. Another danger arises from the fact that nutritive disturbances may occur in the intussusceptum, in consequence of compression of the mesenteric vessels—either *inflammatory adhesions and distortions* or *gangrene of the bowel*. Most commonly the ileum with the ileocecal valve in advance is inverted into the colon—*ileocecal invagination*. Under such conditions the invagination of the bowel has a tendency to increase gradually in size in consequence of the pressure exerted by the column of fecal matter, and in this way the flexures of the colon may be obliterated and be represented by a band passing transversely from one to the other iliac fossa. At times the invaginated portion of intestine may protrude a greater or lesser distance from the anus.

In order to indicate by name the situation of an invagination of the bowel the following *nomenclature* has been adopted: Jejunal or iliac invagination indicates that a portion of the jejunum or the ileum has become invaginated into another portion of bowel of the same name. Jejuno-iliac invagination indicates inversion of the jejunum into the ileum. An ileocolic invagination is distinguished from an ileocecal invagination by the fact that in the former the ileum has found its way through the ileocecal valve into the colon, while in the ileocecal invagination the valve with the ileum is inverted.

Individuals dead of invagination of the bowel usually exhibit evidences of **peritonitis**, or of **rupture of the bowel**, or of **septicopyemia.**

Etiology.—*Children* are most commonly attacked by invagination of the bowel, and *boys* more commonly than girls. The disorder develops with especial frequency *between the third and the twelfth month of life.* Both **constipation** and **diarrhea** are named as causes. At times intestinal polypi induce invagination by exerting traction directly on the intestinal wall. Invagination has also been observed in connection with stricture of the bowel, as, for instance, from *carcinoma.* **Injuries of the abdominal walls,** as by a fall or a blow, have also been mentioned as causative factors.

The *mechanical occurrence of invagination of the bowel* has been repeatedly discussed and investigated experimentally. The conditions are clearest in cases in which a tumor, by its weight, has gradually pulled the intestinal wall inward and downward. Under other conditions it seems most probable and most natural to explain the development of invagination of the bowel by conceiving that a portion of intestine engaged in active movement slips into another portion of bowel situated beyond it, and at rest. The slipping of a lower segment of bowel over an upper is an unnatural process. The invagination of a loop of intestine will occur the more readily if the adjacent portion of bowel is paralyzed or constricted by muscular spasm, and accordingly a distinction has been made between *paralytic* and *spastic invagination of the bowel.* The first variety is believed to be the more common.

Symptoms and Diagnosis.—With the onset of invagination of the bowel the patients are as a rule seized with **abdominal pain** so severe as to compel them to cry out. The pain occurs paroxysmally, and is colicky for the succeeding twenty-four or thirty-six hours. In most patients there is also repeated **vomiting.** Soon there appear in addition **alterations in the stools,** which may be thin and contain mucus and blood. Often the **anus** is open in consequence of paralysis of the sphincter, and mucus and bloody fluid constantly trickle out. The **anal orifice appears**

retracted, and the skin in its vicinity is unusually smooth. Soon, however, the diarrhea ceases, and is replaced by obstinate **constipation.**

The diagnosis of invagination of the bowel becomes the more certain if examination of the abdomen discloses the presence of a **tumor**, which is most frequently situated in the umbilical region or the right iliac fossa. This tumor is of oval shape, almost like that of a sausage, with a smooth surface, and at first of inconsiderable tenderness on pressure. It is, further, distinctive that not rarely it gradually changes its position and increases in size, in accordance with the increase in the invagination. At times it extends transversely from one iliac fossa to the other. In consequence of gaseous distention of the intestines and great tension of the abdominal walls, it may be exceedingly difficult after the lapse of a few days to appreciate the presence of a tumor through the abdominal walls, even if all the aid of the usual devices is invoked—examination with warm hands, the knee-elbow position, conversation with the patient during the examination, examination in a warm bath, chloroform-narcosis. Examination of the rectum with the anointed finger should never be omitted, as it is possible at times to feel the invaginated portion of bowel in the rectum itself or in the vicinity. Little diagnostic aid will, as a rule, be yielded by the introduction of a sound into the rectum or by an infusion into the bowel.

The influence exerted by the intestinal alterations upon the general condition is noteworthy. Symptoms of **shock** appear, as manifested by a cold skin, shrunken features, an anxious expression, and a small, accelerated pulse. Under especially favorable conditions invagination of the bowel may undergo spontaneous reduction, or it may be reduced by artificial aid, and all of the morbid manifestations disappear. Much more commonly serious dangers arise, among which symptoms of **ileus** occupy first place. This may be recognized from the presence of insurmountable constipation, the failure of gas to escape from the anus, and the occurrence of vomiting, with evacuation at first of the contents of the stomach, then of bilious intestinal contents, and finally of material of fecal odor and appearance—so-called *fecal vomiting*, *miserere.* Although this condition alone is indicative of serious menace to life, this becomes in marked degree greater from the development within a short time of *peritonitis*, originating at the point of intussusception, and soon extending to the entire peritoneum. Progressive distention of the abdomen, especially severe pain on palpation of the abdomen, here and there developing dulness on percussion of the abdominal wall, in correspondence with disseminated accumulations of exudate between adherent loops of bowel, usually permit ready recognition of the process. At times **rupture of the bowel** takes place from laceration of the gangrenous intestine. An already existing inflammation of the peritoneum becomes in consequence transformed into a fecal and putrid peritonitis.

At times natural recovery takes place from invagination of the bowel by gangrenous exfoliation of the invaginated portion of intestine, and evacuation with the stools, in consequence of which the previously occluded intestine becomes again patulous. Most frequently spontaneous separation of the gangrenous bowel takes place between the eleventh and the twenty-first day. It is usually revealed by the occurrence of stools of extremely offensive odor and often of bloody character, in which black, necrotic shreds and at times also portions of bowel of considerable length are encountered. Further, the separation of the gangrenous portion of the bowel may be attended with serious dangers, among which uncontrollable *hemorrhage* from and *perforation of the bowel* may be mentioned. At times also *septicopyemia* is superadded in consequence of absorption of gangrenous material, and as a result of which death takes place. Attention should be called here to an important sequel of invagination of the bowel. Should, subsequently, considerable contraction of the cicatricial tissue take place at the point of separation, *stricture of the bowel* and even *occlusion of the bowel* may result, and in consequence of which death may take place.

The *course* and the *duration* of invagination of the bowel may be peracute, acute, subacute, or chronic, accordingly as a few hours, days, weeks, or months, or even years transpire. In the first event death results amid symptoms of shock. It is noteworthy, further, that intestinal invagination exhibits a great tendency to *recurrence*. It is often possible to relieve the invagination, but it recurs within a few hours. The cases are rare in which an invaginated portion of bowel has protruded persistently from the rectum, and has required operative intervention for its removal.

Prognosis.—Invagination of the bowel always demands a serious prognosis, but the probabilities of a favorable issue have become materially improved of late since it has been learned that some of the dangers can be averted by operation.

Treatment.—It is an almost obvious indication that persons with invagination of the bowel should remain constantly in bed, and partake in small amount of exclusively liquid nourishment (milk, milk and coffee, meat-broth), a teaspoonful or tablespoonful at intervals of half an hour. Besides, a hot cataplasm should be applied to the abdomen, and opium be administered internally in order to relieve the pain, and to keep the bowel at rest, thereby avoiding the occurrence of inflammation and extension of the inflammatory process. It should, however, be borne in mind that children, and especially infants, are exceedingly susceptible to the action of opium, so that even small doses may at times be followed by serious symptoms of intoxication. Instead of opium morphin may be employed subcutaneously. Then an attempt should be made to reduce the invagination by **mechanical means.** For this purpose *intestinal infusions of cold water* are to be preferred, and these, if necessary, may be repeated thrice or four times during the day. In order that the water introduced may reach as high a point in the bowel as possible, the patient should occupy the knee-elbow posture or the pelvis should be elevated. Rectal injections in chloroform-narcosis may also be practised. At times the invagination may be observed to correct itself with a distinctly audible murmur. The employment of *injections of air* or of *carbon dioxid* is less convenient and in my opinion less efficient than that of injections of water into the bowel. Injections of air are made most simply with the aid of a rectal tube, connected with the bulb of a Richardson spray-apparatus. In cases in which the invagination has advanced to the rectum an attempt may be made to effect reduction by means of a long but not too rigid *rectal bougie* (sponge-bougie). If recurrences of the invagination take place, it may become necessary to permit the sound to remain in the rectum, fastening it externally.

Attempts have also been made to reduce invagination of the bowel by means of *abdominal massage,* but this procedure cannot be considered as entirely free from danger.

If all efforts have been unattended with success, **surgical treatment** becomes necessary. It is particularly important not to defer this too long, for if necrotic alterations have already taken place in the intestines, or if extensive peritonitis has developed, the results of operation may also be doubtful. The operative procedure will depend upon the existing conditions. Opening of the abdomen (celiotomy), determination of the situation of the invagination, and reduction by traction upon the bowel will first have to be considered. At times loops of bowel have been fastened by means of sutures in order to prevent recurrence. If, however, the invaginated portions of bowel have already become so adherent that separation is not possible, only two procedures remain feasible, namely, either *resection of the invaginated portion of bowel* or the establishment of an *intestinal fistula* above the point of invagination. In the latter event there would still be danger that gangrene might develop at the point of invagination, with its serious consequences.

STENOSIS AND OBSTRUCTION OF THE BOWEL (ENTEROSTENOSIS AND ILEUS).

Etiology.—The causes of stenosis or of obstruction of the bowel may be **congenital** or **acquired.** *Congenital obstruction of the bowel* is rare, and occurs when the anus is occluded or when in the upper portion of the bowel deficient development and closure in certain portions of the intestine have taken place. Death will occur soon after birth, unless in the presence of congenital atresia of the anus the lumen of the intestinal tube can be rendered patulous by means of an operation. *Acquired stenosis* and *obstruction of the bowel* are divided into *intra-intestinal*, *extra-intestinal*, and *parietal* varieties, in accordance with the *situation of the causative conditions*. Among *intra-intestinal causative conditions fecal accumulation*—*coprostasis*—occupies first place. Such a condition may be induced by the presence of *foreign bodies in the intestine*. At times the foreign bodies may have been swallowed (bone, tendon, fibrous remains of asparagus, fruit-kernels, masses of shellac, etc.); at other times they may have been introduced into the rectum through the anus, as, for instance, bottles; and at still other times they may consist of substances from viscera whose excretory ducts discharge into the intestine. The last category includes principally *gall-stones*, which either have become awkwardly impacted in the lumen of the bowel or cause obstruction by cohesion. There are on record a number of instances in which *masses of spool-worms* have obstructed the lumen of the bowel. Rarely *intestinal concretions*—*enteroliths*—act as the cause of obstruction of the bowel.

Among the *parietal causes* of stenosis and obstruction of the bowel *cicatrices* and *neoplasms* may first be mentioned. Not all ulcerative and cicatricial processes lead with the same degree of frequency to stenosis of the bowel. Such a condition occurs but rarely in association with catarrhal and typhoid ulceration; but somewhat more frequently in the case of tuberculous ulcers; still more dangerous are dysenteric and syphilitic cicatrices. Among intestinal neoplasms carcinomata should first be mentioned; there may also be sarcomata, polypi, at times also lipomata and adenomata. Stenosis of the bowel may be due to *hemorrhoids*. Rarely it occurs as a result of *echinococcus* of the intestinal wall. *Displacements of the bowel* often give rise to stenosis or obstruction. The importance of *invagination of the bowel* has already been discussed in the preceding section. *Incarcerated hernia* deserves especial consideration by reason of the frequency of its occurrence. The incarceration may involve an external or an internal hernia. In the latter event either loops of bowel enter abnormal fissures, such as are known to occur in the mesentery and the omentum, and also in the round ligament of the uterus; or, in consequence of previous peritonitis, bands or adhesions have formed between the abdominal viscera and the abdominal wall, beneath which loops of bowel find their way and are incarcerated. There are besides a considerable number of internal hernial openings, in which loops of bowel may be incarcerated. Among these varieties of hernia may be mentioned the duodenojejunal, intra-epiploic, cecal, intersigmoid, iliacosubfascial, anterior retroperitoneal, hernia into the vaginal tunic of the testicle and the broad ligament of the uterus, and diaphragmatic hernia. The last may be congenital or be acquired through traumatism.

An especial variety of obstruction of the bowel results from *twisting* of the intestine (*volvulus*), *axial rotation* of the bowel, and the *formation of a knot*. The longer and the more mobile the mesentery of a portion of bowel the more readily, naturally, will twisting and axial rotation take place. Such conditions develop with especial frequency at the sigmoid flexure of the descending colon, as this part of the bowel turns upon the axis of its mesentery, which, while long, is narrow at its attachment. At times several loops of bowel surround others in the form of a knot, and thus obstruct the lumen of the latter. Most commonly the sigmoid flexure is twisted about dependent loops of ileum. A diverticulum of Meckel or the vermiform appendix may form a knot-like constriction around adjacent loops of bowel and render these impermeable. Naturally, it may happen that such a diverticulum or the vermiform appendix has become adherent with its free extremity to the abdominal wall or to other abdominal viscera, and that loops of intestine enter the interval and become obstructed. So-called *paralytic ileus* should also be mentioned here; its production is dependent upon the fact that the musculature of the intestine is paralyzed throughout a limited extent, and is therefore incapable of propelling the intestinal contents onward. This condition has been observed to occur in association with chronic diseases of the brain and the spinal cord, with peritonitis and perityphlitis, and after celiotomy and the replacement of incarcerated hernia.

Among the *extra-intestinal causes of stenosis* and *obstruction of the bowel* may be included all constrictions due to pressure, and which are usually dependent upon diseases of the adjacent viscera. Naturally loops of intestine may, by reason of fecal accumulation or of the presence of a neoplasm, exert mutual pressure on one another, and thus cause obstruction of the bowel. Tumors and displacements of the liver, spleen, or kidney, marked increase in the size of the pancreas, neoplasms of the omentum, the uterus, the ovaries, and the bladder, parametritic, perimetritic, and peritonitic exudates, the impregnated and retrodisplaced uterus, these all are capable of exerting pressure upon the intestine. At times stenosis of the bowel has been observed in cases of prostatic hypertrophy, and in consequence of the presence of uterine pessaries. The disorder occurs most commonly in *men*. It may develop at any time of life, although age has a certain influence upon the nature of the causative factors. Thus, invagination of the bowels occurs with especial frequency in childhood, and carcinoma of the intestine, on the other hand, in the aged.

Anatomic Alterations.—The bodies of those dead from stenosis or obstruction of the bowel are characterized by unusual

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Diagnosis.—The *symptoms of obstructio*
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lindric masses occur, presenting the appear

ance of coffee-beans, or the fecal discharges of the sheep or the goat. The same variety of feces occurs also in cases of inanition and of constipation when the latter is dependent upon spasm of the musculature of the bowel. The feces may also exhibit gutter-like depressions, as, for instance, when intestinal polypi are present. If the fecal matter be forced through the constricted area, most severe **abdominal pain** is often produced, which may cause syncope and general clonic muscular spasm. Although constipation is the rule in cases of constriction of the bowel, **diarrhea** is by no means rare. This may result from the maintenance by the stagnating fecal material of a chronic catarrh of the intestinal mucous membrane above the point of constriction. It is noteworthy that the thin stools often contain unusually hard masses of fecal matter. At times **blood** is found **in the stools**, and its appearance is to be explained by mechanical injuries inflicted by the fecal matter, and in other instances by tissue-disintegration.

In order to determine the *seat of constriction* of the bowel consideration should be given to the *degree of abdominal distention*, for the more deeply situated the constriction the greater will be the abdominal distention. If *intestinal movements* are visible, the point at which these are especially active and regularly terminate is often of diagnostic importance. As a matter of course, an effort should be made by careful *palpation* through the *abdominal walls*, the *vagina*, and the *rectum*, to reach the obstruction directly. Examination of the rectum with a sound, and an *injection into the bowel*, as well as the employment of a *rectal speculum*, will also yield valuable diagnostic information. A careful *history* and a knowledge of the *mode of development* of the disorder are also important. Thus, for instance, attention should be directed to preceding dysentery and syphilis, to tuberculous alterations, to the state of the bowels, to sudden or gradual development, and the like. In this way the exciting cause of the disorder, and from this not rarely also its seat, can be determined.

The *course of stenosis of the bowel* may be terminated within a few hours, or it may be extended over years, in accordance with the nature of the causative factors and the care observed by the patient with regard to diet and to regularity in movement of the bowels. One of the most important dangers consists in the fact that the constriction may progress to occlusion of the bowel. This may develop gradually or occur suddenly, as, for instance, from the ingestion of indigestible articles of food, which may accumulate above the point of constriction like a valve, or from constipation. Naturally most cases of *obstruction of the bowel* or *ileus* begin suddenly. Not rarely heavy lifting, the ingestion of excessively cold or of indigestible food, a fall or a blow upon the abdomen, and the like are assigned as causes. At times the disorder sets in with severe pain in the abdomen.

The principal symptoms of obstruction of the bowel are ***retention of fecal matter and of intestinal gases***, and ***fecal vomiting—miserere.*** Naturally, it may happen at the beginning of the disease that repeated thin stools are voided, and these, in cases of invagination of the bowel, may partake of a mucous and bloody character, but soon thereafter most obstinate *constipation* sets in. Like the feces, gas also may be unable to pass the obstructed portion of bowel, and the patient cannot discharge *flatus*. Naturally, if air is introduced into the bowel in addition to fluid in the process of making an intestinal infusion, it may happen that the air is expelled in the form of flatus, a phenomenon without significance that should not be referred to a restoration of the permeability of the bowel. The *vomited matters* consist at first only of gastric contents, and accordingly their appearance varies with accidental conditions. After the stomach has been emptied the greenish bilious contents of the small intestine are voided in the act of vomiting. Finally, vomiting of fecal matter occurs. This is conspicuous, in the first place, from its marked fecal odor. Usually it consists of a yellowish, thin mass, which often contains light-yellow or brownish-yellow fragments of fecal matter. The presence of formed fecal nodules is an exceptional occurrence. The deeper the situation of obstruction of the bowel, the more abundant and the more fecal does the vomited matter become, for the greater will be the contents of the intestine, and the fecal matter acquires its distinctive character in the deeper portions of the bowel.

In the past various explanations were offered for the *occurrence of fecal vomiting*. At present it is assumed not that it is dependent upon antiperistaltic intestinal movement, but that the intestinal contents endeavor to escape backward from the obstruction in consequence of the pressure resulting from the peristaltic intestinal movement.

On examination *distention of the abdomen, borborygmi*, and *active peristaltic intestinal movements* are observed for the same reasons as in cases of intestinal constriction. Not rarely certain loops of intestine appear as unusually thick and tensely distended structures lying one upon another. Careful palpation of the abdomen, particularly of all external hernial orifices, and thorough examination through the vagina and the rectum should never be omitted, in order to determine, if possible, the situation and possibly also the nature of the obstruction. The deleterious influence of intestinal obstruction upon the *general condition* is most striking. The features of the patient soon become sunken, and he often speaks in a faint and high-pitched voice, like a cholera-patient. The bodily temperature is frequently subnormal, and the pulse small and running. After severe vomiting the skin loses its turgor, remains elevated in folds, and feels moist and cold like that of an amphibious animal. The *urine* is diminished in amount and dark in color. Examination of the urine for *indican* is of importance, for if peritonitis be absent and carcinoma is not the cause of the obstruction, increased elimination of indican is indicative of obstruction of the small intestine.

For the demonstration of indican the test of Jaffe may be employed. To this end a test-tube is half-filled with urine, the remaining half with hydrochloric acid, and from 1 to 5 drops of fresh concentrated chlorinated lime are added. After the addition of each drop the opening of the tube should be closed with the thumb, and the tube inverted several times. The addition of chlorinated-lime solution is suspended when a deep-blue discoloration of the urine no longer takes place. Chlorinated lime in excess would cause a diminution in the intensity of the color. An increase in the amount of indican in the urine will be indicated by the fact that the urine acquires a violet-red, bluish, or deep blue-black color. Obstruction of the small intestine gives rise to an increase in the amount of indican in the urine in consequence of interference with intestinal digestion and increased fermentation of proteid bodies in the contents of the bowel. The substances of the indigo-group are derived from proteid decomposition in the intestinal contents. For the same reason the urine contains an abundance of *phenol* and *ethereal sulphates*.

The *duration of obstruction of the bowel* may at times extend over only a few hours, death occurring amid manifestations of *shock*. In other instances the disorder persists for days, and even for more than a week. Death results in consequence of either *progressive exhaustion* or *complications*, of which *peritonitis* and *rupture of the bowel with secondary perforative peritonitis* are the most common. Not rarely *inspiration-pneumonia* develops, becoming converted into *pulmonary abscess* and *gangrene of the lung*, and resulting from the aspiration of vomited matters. The condition is often unrecognized during life, for the reason that frequent raising of the patient to the erect posture for the examination of the posterior portion of the lungs is avoided on account of weakness. In some cases rupture of the bowel takes place into an encapsulated cavity, and there forms a fecal abscess, with which *septicopyemia* may be associated. It may also happen that after previous adhesion of the intestine with the abdominal walls rupture takes place externally, with the formation of a *fecal fistula*. A sort of *natural cure* takes place in rare instances from the formation of adhesions between loops of intestine above and below the point of obstruction, with the establishment of communication through a fistula—so-called *bimucous intestinal fistula*. Relief from ileus may be recognized from the fact that flatus again escapes, bowel-movements are resumed, and the vomiting ceases. The escape of flatus is of especial importance, as the residue of fecal matter below the point of constriction might give rise to a bowel-movement. Some persons pass through *several attacks of intestinal obstruction* in the course of their lives. At times the causative factors are obvious (fecal accumulation, gall-stones, incarcerated hernia, invagination), while in other instances, on the contrary, it is impossible to discover the causative factors.

Obstruction of the bowel as such is usually easy of *diagnosis*, for *constipation*, *failure of gas to escape from the bowel*, and *fecal vomiting* are readily recognizable and reliable symptoms. On the other hand, insurmountable difficulties often arise with regard to the determination of the nature and the seat of the obstruction. *In the presence of symptoms of obstruction of the bowel careful examination of all hernial openings for incarcerated hernia should never be omitted.* I have knowledge of many instances in which even so-called first authorities in the domain of internal medicine had neglected this rule. A harmless operation for hernia at the proper time would have saved the patient, who otherwise dies subsequently in consequence of gangrene of the incarcerated loop of bowel, peritonitis, and septicemia. If an external incarcerated hernia can be excluded, it will be possible in some cases to form an opinion as to the anatomic nature of the obstruction from the history (previous constipation, gall-stones, tuberculosis, syphilis, dysentery, previous peritonitis, etc.). The *age* of the patient is deserving of consideration, as sudden obstruction of the bowel in children is frequently dependent upon invagination and in the aged upon carcinoma. In cases of intestinal invagination the mucous and bloody stools at the onset of the disease are of diagnostic importance. *Palpation of the abdomen* and *examination of the abdominal cavity through the vagina and the rectum* may also furnish valuable diagnostic information (a longitudinal tumor in cases of invagination, cicatricial stenosis or nodular carcinomatous new-growth in the rectum, retrodisplacement of the uterus, and the like). In other instances, whether axial rotation, knot-formation, or internal strangulation of the bowel is present can scarcely be determined with any degree of probability during life. The differentiation between paralytic and mechanical ileus in consequence of peritonitis and perityphlitis remains as yet among the unsolvable problems.

In the recognition of the *seat of obstruction* of the bowel the *local intestinal condition* is important (palpable constriction in the rectum, palpable tumors, increased peristaltic movement only to a certain portion of the bowel); besides, considerable importance is to be attached to the *amount of indican in the urine*, for if this be increased it indicates an obstruction of the small intestine, if peritonitis be not present and carcinoma can be excluded. Less reliable from the diagnostic standpoint is *distention of the abdomen*, although it is true that this is the greater the deeper the seat of the obstruction in the bowel.

In those rare cases in which, especially at the onset of obstruction of the bowel, severe diarrhea and vomiting occur, error has at times been committed in confounding the condition with *Asiatic cholera* and *poisoning* (arsenic, antimony). The diagnosis of cholera can at present be made bacteriologically, and in cases of poisoning the history is of importance.

Prognosis.—The prognosis of stenosis and obstruction of the bowel is unfavorable when the causative conditions are irremediable. Even under other conditions also, however, it is grave.

Treatment.—**Prophylactic measures** are indicated especially in the presence of stenosis of the bowel. The *diet* should be so regulated that neither the fecal masses should be too large nor constipation should arise. Besides, obstructions that are removable should be corrected, as, for instance, stricture of the rectum—an indication that is included in the *causal treatment*. If symptoms of intestinal obstruction have appeared, **rest in bed, a liquid diet, and opium** should be prescribed:

℞ Powdered opium,	0.02 (⅓ grain);
Sugar,	0.3 (4½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

In the second place, **causal treatment** should be carried out. Strangulated external hernia should be replaced or released by abdominal section. The clinical picture of intestinal obstruction may persist after reposition, either because, in replacement *en masse*, actual release of the constriction has not been effected, or because the incarcerated loop of intestine has been paralyzed, with resulting paralytic ileus. Obstruction of the bowel in consequence of fecal accumulation can be relieved by irrigation of the bowel with ice-water, repeated at intervals of two hours. The first injections are frequently without effect, the fluid returning clear, because the lowermost fecal matters are of stony hardness and must first be softened. At times it even becomes necessary first to remove a number of fecal masses from the rectum with the fingers before the column of fecal matter, situated higher up, can escape, sometimes in remarkable amount. Foreign bodies in the rectum, also, must be removed with the fingers, etc. Should there be no indication for causal treatment—as, for instance, if the condition causative of the obstruction be not recognized—attempts should be made to restore the permeability of the bowel by **intestinal injections of cold water**, repeated several times daily. Instead of injections of water, **enemata** of air or of **carbon dioxid** have also been employed. In the presence of severe fecal vomiting the stomach should be washed out several times, as the symptoms of intestinal obstruction are said to have disappeared after **irrigation of the stomach**. By some, **mercury** in tablespoonful-doses up to 1000 grams has been successfully employed. In a patient with ileus, operation on whom had already been decided upon, I observed the bowels to be moved and recovery to take place after all other measures had been attempted without success, as a result of the application of one pole of the **faradic current** in the rectum, while the other was placed upon the abdominal wall.

Actually certain relief can be expected, however, only from **surgical intervention**; but this should not be deferred until inflammation and gangrene of the obstructed portion of bowel or general peritonitis has developed. One plan to be pursued consists in opening the abdomen, finding and removing the obstruction, and freeing the bowel, while another consists in the establishment of an intestinal fistula above the point of occlusion. Further, the search for an obstruction is often extremely difficult on account of the great distention of the intestine with gas. If it be impossible to find the obstruction, one will be compelled to be satisfied with the establishment of a preternatural anus. This, however, will by no means remove all sources of danger, as inflammation and gangrene of the obstructed portion of bowel may yet result and give rise to general peritonitis. At times, further, the bowel has been observed to become again patulous a short time after the establishment of a fecal fistula, so that subsequently it became possible to close the fistula. Clinicians have stated that sometimes after *puncture of the bowel* the symptoms of intestinal obstruction disappear; but surgeons will probably not look favorably upon this kind of intestinal surgery—and properly so, in our opinion.

HEMORRHOIDS.

Etiology.—Hemorrhoids consist in *varicose dilatation of the hemorrhoidal veins.* They occur with extreme frequency, because stasis of blood in the hemorrhoidal veins is greatly favored by the dependent position of the veins and their freedom from valves. The observation that hemorrhoids are common in some families and develop under such conditions upon slight provocation, appears to indicate that at times a *congenital want of resistance in the walls of the veins* is also probably operative. The *causes of the stasis* are at times **local** and at other times **general**. Among the local causes may be included *constipation, carcinoma of the rectum, chronic proctitis, prostatic hypertrophy, retrodisplacement of the uterus, neoplasms of the uterus*, and *pregnancy*. *Long-continued sitting* and *riding* also favor the development of hemorrhoids. The disorder is therefore encountered with particular frequency in certain classes, as, for instance, students, shoemakers, cavalrymen. Not rarely the affection develops in persons who indulge in *sexual excesses*, or preferably sit upon *upholstered furniture*, probably in consequence of diversion of an excessive amount of blood to the sexual organs and their vicinity. Hemorrhoids are a frequent sequel of *stasis in the portal vein*, and they develop often therefore in cases of thrombosis of this vein, cirrhosis of the liver, and also other diseases of this organ. Frequently they depend upon *gen-*

eral venous stasis. They occur, therefore, in cases of chronic disease of the heart and of the respiratory organs. I have observed them also in a number of instances in persons who play upon wind-instruments, in glass-blowers, and in those who carry heavy loads. *Obese individuals,* who are inclined to indulge excessively in the pleasures of the table, often suffer from hemorrhoids.

As a rule, hemorrhoids do not develop before *adult life,* as may be readily understood from the nature of the causative factors. *Men* suffer more commonly than women. The disease is especially common in the Orient, being favored perhaps by sexual excesses.

Anatomic Alterations.—A distinction is made between *external* and *internal hemorrhoids,* accordingly as the venous dilatation is situated without the sphincter ani muscle, and is directly exposed to ocular inspection, or above the sphincter muscle in the lower portion of the rectum. In rare instances the mucous membrane of the descending colon is found covered with bluish-black varices up to the sigmoid flexure of the colon. At times hemorrhoids form single **nodules** from the size of a pea to that of a walnut, and even that of an apple. Sometimes such nodules are disposed close together in a circular manner. At times adjacent nodules have coalesced, and have formed a sort of venous *cavernoma.* Further, hemorrhoidal dilatations diminish greatly in size as a rule after death. The adjacent **mucous membrane of the rectum** is generally in a state of chronic catarrh, with marked thickening of the submucosa. At times thrombi have formed in some of the venous dilatations, and these subsequently undergo calcification. These bodies of stony hardness are designated **vein-stones or phleboliths.**

Symptoms and Diagnosis.—Many persons believe that they suffer from hemorrhoids when these are not present, while others have hemorrhoids, but are free from symptoms and without intimation of the existence of the disorder. The single certain symptom is the **demonstration of hemorrhoidal nodules,** which, if they be external, can be seen directly on inspection of the anus to consist of more or less tensely distended bluish nodules of varied size, and if they be internal can be felt by means of the finger introduced into the rectum as soft nodular elevations. Digital examination of the rectum should never be omitted in order to determine positively whether internal hemorrhoids exist at all, or internal hemorrhoids are present together with external hemorrhoids. The attention of some patients is first directed to the presence of this disorder by the occurrence of *hemorrhoidal bleeding,* and this also is frequently observed only accidentally from the fact that the paper used for cleansing the anal region after a bowel-movement feels slippery and on examination is found to be streaked with blood. When the hemorrhage is more copious the body-linen and the bed-linen may be saturated with blood. The occurrence of the hemorrhage is not rarely preceded by peculiar manifestations, which were formerly designated as **hemorrhoidal molimina.** Among these are a sense of fulness of the head, of pulsation in the head, headache, vertigo, palpitation of the heart, a sense of fear and oppression, a sensation of tickling or of tension at the anus, a sense of beating in the anal region, and the like. These disturbances generally cease as soon as the hemorrhage has begun, and the majority of patients become conscious of a sense of relief and freshness after the termination of the bleeding. They anticipate with satisfaction the occurrence of such a hemorrhage, which they consider as a beneficial discharge of impure fluids, and for this reason the designation "golden vein" was formerly employed instead of hemorrhoids. When opportunity is afforded for examining daily patients with external hemorrhoids it will frequently be found that a hemorrhoidal nodule progressively increases in size. The tension of the overlying skin becomes increased, the skin becomes thinned, and finally it and the wall of the vein rupture, and blood is discharged externally. The statement that the hemorrhages occur at definite intervals, as does menstruation, is incorrect. Often no cause for the occurrence is apparent, while at other times it has been preceded by indulgence in alcohol or sexual excitement, persistent sitting, or excessive walking and riding. The *amount of blood lost* varies exceedingly. It is of exceptional rarity that the hemorrhage is so profuse and uncontrollable that life appears to be threatened. Profound anemic states, with their varied disorders, especially palpitation of the heart and dyspnea, do, however, occur with frequency, and require treatment. Mucous discharges from the anus that sometimes take place with the evacuation of fecal matter or gas have been described as *blind* or *mucous hemorrhoids*. These are, however, merely the manifestations of a chronic catarrh of the rectum. At times they constitute the only symptom in patients with hemorrhoids. While it is true that such patients are often depressed and hypochondriacal, such a condition is to be attributed to the chronic catarrh of the rectum.

Patients suffering from hemorrhoids often complain of **paresthesia in the anal region,** especially of tickling and burning. There is also often a sense of a foreign body in the rectum, particularly in walking. Not rarely **constipation** exists, and it is necessary in each case to determine whether this be a cause or a consequence of the hemorrhoids. It happens but rarely that hemorrhoidal nodules attain such a considerable size as to give rise to *constriction* or *obstruction of the bowel.* Internal hemorrhoids at times protrude from the anus, particularly with movements of the bowel, but cannot be returned because they are incarcerated by the sphincter ani muscle. Such *strangulation of hemorrhoidal nodules* gives rise to intense pain in the anal region, so that even strong individuals writhe and groan with pain. Usually they occupy the lateral decubitus, with the lower extremities flexed at the hip and the knee. If the incarceration be not speedily removed, inflammation and gangrene of the strangulated nodules take place, and consequent absorption of the exciting agents of inflammation into the blood-stream may give rise to *septicopyemia*. *Inflammation of hemorrhoidal nodules* also occurs at times independently of strangulation. Under such conditions the inflammatory process not rarely extends to the periproctal connective tissue, with the development of a *periproctal abscess*. Accordingly as this ruptures externally or internally into the rectum, or in both directions at the same time, an external, an internal, or a complete *rectal fistula* develops. Hemorrhoids also occasion a predisposition to *fissures* or *tears of the anus*. Chronic *eczema of the skin*, likewise, develops not rarely in the neighborhood of the anus.

Hemorrhoids may undergo complete involution if the causative conditions are removed, as, for instance, in the case of pregnancy. Often, however, the causative conditions are incurable, so that the disorder may persist throughout the whole of life and exhibit only frequent variations in course. Although the diagnosis of hemorrhoids is easy, it may be equally difficult to determine the causative conditions. In persons of advanced years digital examination of the rectum for carcinoma should never be omitted, as this condition frequently causes hemorrhoids, and even independently of these may give rise to mucous and bloody discharges from the rectum, which by a careless diagnostician might be at once attributed to insignificant and harmless hemorrhoids. Should there be any doubt as to the nature of outgrowths at the anus (condyloma, folds of skin, epithelial carcinoma), puncture with a sterilized needle should be practised, and the escape of a few drops of blood looked for.

Prognosis.—Hemorrhoids constitute an annoying rather than a dangerous condition. The likelihood of cure depends upon the curability of the causative factors.

Treatment.—In the treatment of hemorrhoids causal therapy will first require careful consideration. Persons suffering from hemorrhoids should be especially careful to secure regular evacuation of the bowels and to cleanse thoroughly the anal region after each bowel-movement. Active pedestrianism and the use of hard chairs in place of upholstered furniture should be recommended. Sexual and alcoholic excesses should be avoided. If the causative factors be irremediable or if hemorrhoids cause distress by reason of their size or of hemorrhage, or of incarceration and inflammation, they can only be removed by surgical operation, although the patient must be informed that they may recur after the lapse of some time. Operations for hemorrhoids were formerly looked upon with doubt justly on account of the frequent occurrence of septicopyemia.

Various procedures have been recommended; for instance, ligation and clamping of the venous nodule, removal by means of the galvanocautery, excision of the nodule, dilatation of the anus, and injection of carbol-glycerin. Among the complications *hemorrhoidal bleeding* does not, as a rule, require especial intervention. If *incarceration of hemorrhoidal nodules occur,* an effort should be made to return them into the rectum by means of gradual pressure exerted with the aid of a clean pad dipped in oil. Should this fail they should be covered with a tepid solution of mercuric chlorid (1 : 1000) and their exfoliation awaited. The treatment last named is applicable also in cases of *inflammation of hemorrhoidal nodules* and of the vicinity. *Periproctal abscesses* require incision with the knife.

MELENA OF THE NEWBORN.

The occurrence of *bloody vomiting* or *hematemesis,* and *of intestinal hemorrhage* or *enterorrhagia,* in the newborn is known as melæna neonatorum. At times the condition begins with hematemesis, and at other times with enterorrhagia. It may also happen that perhaps only the one or the other phenomenon takes place. The disorder is rare, but is attended with great danger of death from hemorrhage and exhaustion. As may be understood, the condition can be readily recognized from its conspicuous symptoms, but care must be observed to avoid confounding it with the presence of blood that has been swallowed and has gained entrance into the gastro-intestinal canal of the infant during birth, or from injured nipples. *Causative factors* can at times not be elicited. Sometimes parturition has been abnormal, or there has been some peculiarity in the ligation of the umbilical cord. In some cases the condition appears to be a sequel of septic infection. Recently melena of the newborn has been attributed variously to injuries of and hemorrhage into the brain and spinal cord. As a rule, *ulceration* of the gastro-intestinal mucous membrane is found, but nothing definite is known as to the origin of this. It has been attributed to embolic, thrombotic, and mycotic influences, but these are all undemonstrated hypotheses. Therapeutically *cold fomentations* may be applied to the abdomen and Malaga wine administered in drop-doses to avert exhaustion.

ENTEROPTOSIS.

Displacement of the bowel preferably involves the colon, especially its transverse portion, whose loops may thus at times descend into the true pelvis. As a result there naturally is difficulty in the propulsion of the intestinal contents, and the patients complain of constipation and digestive disturbances of varied kind. The condition can be recognized by distending the large intestine with air or large amounts of water through the anus, and practising percussion along the course of the transverse colon. Enteroptosis is usually found in association with displacement of other viscera, and particularly wandering kidney, both conditions being dependent upon the same causes. The patients are frequently pallid and nervous individuals.

The **treatment** consists in improvement of the general nutrition, abdominal massage, the wearing of an abdominal binder, care to secure a daily evacuation of the bowels, and the correction of displacement of other abdominal viscera, such as a wandering kidney.

INTESTINAL NEUROSES.

Less is known concerning intestinal neuroses than concerning gastric neuroses. The former may at times be motor, at other times secretory, sensory, or vasomotor. Mixed intestinal neuroses also occur.

MOTOR INTESTINAL NEUROSES.

ATONY OF THE INTESTINE.

Etiology.—Atony of the intestine may also be designated *relaxation of the muscular coat of the intestine.* In consequence, the bowel is incapable of properly propelling its contents and *constipation* results. As the latter not rarely persists throughout life, the condition has also been designated habitual constipation. Often the disorder is **hereditary** and **congenital**, and possesses the characters of a familial disease. In other cases the disease is **acquired** in consequence of previous chlorosis, debilitating diseases, and central neuroses (hysteria, neurasthenia, hypochondriasis). Neglect of intestinal activity also is followed by atony of the bowel. The condition is therefore frequently observed in women who are in the habit of suppressing the desire for stool in order not to be interrupted in their work. The disorder is exceedingly common, as the modern mode of life favors its development in marked degree. It is not rarely observed in pupils at school.

Symptoms, Diagnosis, and Prognosis.—Atony of the bowel gives rise to **constipation**, so that the patients at times have a bowel-movement once or twice a week, or not at all without the employment of purgatives. At the same time it is characteristic that symptoms are wanting indicative of anatomic changes in the bowel. When the bowels are moved, excessive expulsive efforts must be made, and the feces are dry, hard, and black, as if charred or burned, and they readily cause severe pain in the rectum and the anus by mechanical irritation. Injuries of the mucous membrane and (generally small) hemorrhages may also occur. The longer the interval between the bowel-movements the greater becomes the complaint of abdominal distention, disagreeable pressure, and even of pain in the abdominal cavity, and the like. The constipation has, without doubt, an injurious influence upon the *general nutrition*, and many patients are extremely pallid, and become emaciated, depressed, and incapable of mental activity. Possibly these may be consequences of auto-intoxication, with evidences of increased intestinal putrefaction. The disorder is not dangerous to life, but is distressing and obstinate.

Treatment.—The treatment is in part **general** and in part **local**. The *general treatment* should be directed to the correction of existing anemia, conditions of weakness, and neuroses. The *local treatment* should secure regular evacuation of the bowels, and the same remedies will be required that have already been mentioned in the treatment of constipation in consequence of chronic intestinal catarrh (pp. 256 and 257).

NERVOUS DIARRHEA.

The nervous system exerts an important influence upon intestinal activity, as many persons are seized with a desire for stool and looseness of the bowels under the influence of fear and embarrassment. In hysterical and neurasthenic individuals diarrhea not rarely occurs for a considerable length of time, and with frequent relapses, but with an absence of all signs of anatomic changes in the bowel. *Tabetic patients* also are at times seized with attacks of nervous diarrhea. It is important to appreciate the disorder properly from its etiologic aspect, for the *treatment of the general condition* and the *prescription of bromids* are far more serviceable than the employment of styptics. Nervous diarrhea may be due to increased peristaltic activity of the intestine alone, although it is not impossible that at times increased secretion of intestinal juice also takes place.

PERISTALTIC INTESTINAL UNREST (NERVOUS INTESTINAL TORMINA).

Like the stomach (p. 236), the intestine also may be the seat of abnormally excessive motility, which gives rise to a disagreeable feeling of rumbling and of unrest, and disturbs sleep at night. The causative factors and the treatment are identical with those of nervous unrest of the stomach.

SENSORY INTESTINAL NEUROSES.

NERVOUS SPASM OF THE INTESTINE.

In hysterical and neurasthenic individuals spasm of the mus-
the intestine sometimes occurs within certain por-
rise to circumscribed abdominal

pain, and also to constipation. There are thus two varieties of constipation due to intestinal neuroses, namely, the atonic and the spastic. The peculiar appearance of the feces is considered distinctive of the latter. As with stenosis of the bowel, the feces form small, abbreviated columns or they resemble coffee-beans, or the feces of the sheep and the goat.

The **treatment** should be directed to the cure of the fundamental disorder. Locally narcotics may be advised, especially bromids, opium, and belladonna.

NERVOUS ENTERALGIA.

Etiology.—Intestinal pain independent of anatomic alterations occurs especially in **anemic, hysterical,** and **nervous individuals.** It is well known that in cases of **tabes dorsalis** attacks of severe intestinal pain not rarely occur—so-called intestinal crises At times **toxic influences** are operative, as a result of which the colic due to lead and copper is best known. **Gouty patients** also are not rarely seized with attacks of intestinal colic. Some persons suffer from intestinal pain after **indulgence in certain kinds of food** without the occurrence of other derangement of intestinal activity **Intestinal worms** also at times give rise to intestinal pain, as do likewise **fecal accumulation** and **gaseous accumulation** in the bowel Intestinal pain of reflex origin at times attends **diseases of the uterus,** the **kidneys,** and the **liver.** Attacks of intense pain are sometimes excited by old **peritonitic adhesions.**

Symptoms and Diagnosis.—The principal symptom of the disorder, which is also known as intestinal colic, is **abdomina pain.** This is most commonly referred to the umbilical region but it may radiate thence for a considerable distance, and at times it changes its position. Its severity is susceptible of great variation, and fluctuates between slight abdominal griping and cutting and stabbing pain of such intensity that the patient writhes in bed, while the skin becomes pale and cool (reflex vascular spasm) cold sweat appears, the face presents an expression of fear, the pulse is small and hard, and the heart-sounds faint. Syncope (cerebral anemia) and general clonic spasm may occur. A times palpitation of the heart, a sense of oppression, contraction of the cremasteric muscle, and the like, may be present. The *duration of an attack of pain* is susceptible of great variation, as is also the *frequency of recurrence.* At times an attack of colic terminates rather suddenly, after vomiting or the escape of gas from the intestine. The **abdomen** is often scaphoid and distended and of board-like hardness, as, for instance, in the presence of lead-colic, although abdominal distention may also result from fecal and gaseous accumulation. As distinctive, in contradistinction from intestinal pain dependent upon anatomic changes in the bowel, it is pointed out that strong pressure upon the abdominal walls does not increase, but usually mitigates the pain, and, as a matter of fact, the patients not rarely press their hands firmly upon the abdomen or apply the abdomen forcibly against some hard body.

Prognosis.—Death scarcely ever occurs as the result of an attack of nervous intestinal pain, and if the fundamental disorder is curable the prospects are good for the prevention of a recurrence of the attacks of pain.

Treatment.—Attacks of nervous intestinal pain are most speedily and most certainly relieved by a **subcutaneous injection of morphin** (0.3 : 10—4.5 grains : 2½ fluidrams; from 0.25 to 0.5—(from 4 to 8 minims—subcutaneously), but this should be resorted to only when the pain has attained a marked degree of severity. Under no conditions should the patient be entrusted with the syringe, morphin, and the injection, because the danger of and the temptation to morphinism are too great. In less severe cases the abdomen should be covered with a **hot cataplasm**, and hot carminative infusions may be drunk (peppermint-leaves, fennel, caraway—1 tablespoonful to 3 cups of hot water for an infusion). Besides, consideration should be given to the causative factors—**causal therapy**—which coincides with the *prophylaxis*.

ANIMAL PARASITES OF THE INTESTINE (INTESTINAL HELMINTHIASIS).

PROTOZOA IN THE INTESTINE.

Protozoa in the intestine have been frequently discovered accidentally on microscopic examination of the stools. They have been found especially in cases of chronic diarrhea, so that it is assumed not without reason that they are capable of inducing or maintaining chronic diarrhea. Many physicians consider these parasites as harmless bodies. Earnest consideration has been given in recent years to the presence of amebæ in cases of tropical endemic dysentery, so that the designation amebic dysentery has been employed.

Stools intended for examination for the presence of protozoa should be fresh, for on standing the protozoa are transformed into roundish, immobile structures, which cannot be distinguished from the colorless blood-corpuscles. It is advisable to obtain the fecal matter directly from the rectum by means of blunt-edged glass tubes.

Among the better known protozoa are the following:

1. *Amœba coli.*—This is a rhizopod, and appears in the form of roundish granular bodies, with a nucleus and a nucleolus, which engage in movement by the extension and retraction of pseudopods (p. 293, Fig. 28). A distinction has further been made between Amœba coli, Amœba coli mitis, Amœba intestinalis vulgaris, and Amœba coli felis.

2. *Cercomonas intestinalis* (p. 294, Fig. 29) is a pear-shaped body, which possesses a long vibratile flagellum at its anterior extremity, and a caudal process posteriorly, which serves as a means of attachment. This body, like all of the following protozoa of the intestine, belongs to the *Infusoria.*

3. *Cercomonas coli* is provided with four vibratile flagella arising from the anterior portion of the body. A lateral border undulates (p. 294, Fig. 30).

4. *Trichomonas intestinalis.*—This parasite resembles an almond-kernel in shape. Cilia are present upon one side, while posteriorly there is a pointed process for purposes of attachment (p. 294, Fig. 31).

5. *Megastomum entericum.*—This parasite has been compared with the half of a pear. Anteriorly it is provided with six flagella, which in the dead parasite have disappeared (p. 294, Fig. 32).

6. *Balantidium s. Paramæcium coli.*—This parasite is egg-shaped and is surrounded by cilia. Anteriorly an oral orifice and posteriorly an anal orifice can be recognized. The interior of the body contains vacuoles and particles of food (p. 294, Fig. 33).

It is well in all cases to cause the expulsion of protozoa from the intestine even when morbid manifestations are not present. Intestinal infusions of quinin (1.0 : 1000), of mercuric chlorid (0.3 : 1000), or of a solution of tannic acid (0.5 per cent.) are especially to be recommended.

WORMS IN THE INTESTINE.

FLAT WORMS (PLATYHELMINTHES).

TAPEWORMS (CESTODES).

Etiology.—Among the various kinds of tapeworm three are of practical significance on account of their frequent and widespread occurrence, namely, the *unarmed tapeworm, Tænia mediocanellata s. saginata,* the *armed tapeworm, Tænia solium,* and the *Bothriocephalus latus.* Tapeworms are acquired only by the ingestion of **measly meat.** In the stomach the capsule of the measle is digested, and its head or rather that of the tapeworm (scolex) is set free, and then develops within the intestine by the formation of segments or proglottides into the mature parasite. From time to time the more distal (the oldest) segments are expelled. Animals that eat feces contaminated with proglottides digest the segments. In this way the contained ova are set free, and migrate from the intestine into the muscles, where they again develop into measles. The *Tænia saginata* is acquired from the *measle of beef,* the *Tænia solium* from that of *swine,* the *Cysticercus cellulosæ* and the *Bothriocephalus latus* from that of *fish.* As the use of beef is customary in all parts of the world, it may be readily understood that the Tænia saginata occurs most commonly and in all countries. The use of pork is avoided by many on account of its fat, and is forbidden Jews and Orientals on religious grounds, so that the Tænia solium is much less common. The occurrence of Bothriocephalus latus is confined to certain situations on the coast and on inland seas, among which may be mentioned the Russian provinces on the Baltic Sea, the coast of Eastern Prussia and Pomerania, and the banks of the Lake of Geneva. Various fish may contain measles, such as salmon, eel-pout, pike, salmon-trout.

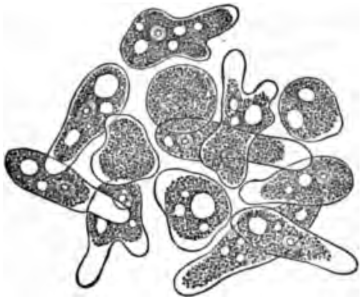


FIG. 28.—Amœba coli (after Lösch).

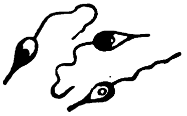


FIG. 29.—Cercomonas intestinalis (after Lambl).

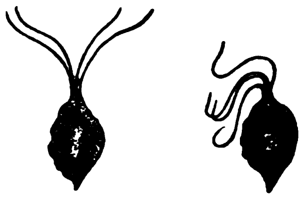


FIG. 30.—Cercomonas coli (after May).

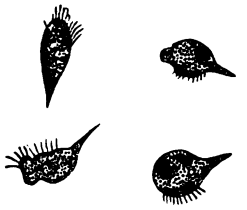


FIG. 31.—Trichomonas intestinalis (after Zenker).

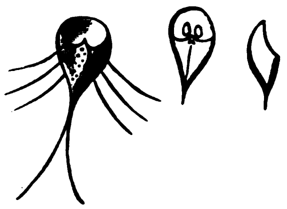


FIG. 32.—Megastomum entericum: *a*, in the fresh, *b*, in the dead state (after Bizzozero and Grassi).

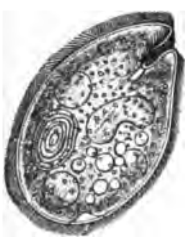


FIG. 33.—Balantidium s. paramœcium coli.

Tapeworms occur in different countries with varying frequency. In the Orient persons free from tapeworms are almost the exception. This may be explained in part by local customs—as, for instance, the use of raw meat favors the prevalence of tapeworm, as the measles are generally destroyed and rendered harmless by cooking; and in part by want of cleanliness in the stables, and carelessness in the inspection and sale of meat. If domestic animals are not restrained from eating human fecal matter, if the sale of measly meat is not prohibited, if measly meat is sold in butcher-shops together with healthy meat, and if all meat is carved with the same knife, all of these conditions are factors that favor the infection of animals and human beings. Tapeworms occur with greatest frequency in adults. They have been variously observed in children when raw shaved beef has been recommended in the presence of diarrhea.

Symptoms, Diagnosis, and Anatomic Alterations.—The presence of a tapeworm in the intestine can be recognized with certainty only when **proglottides** or **ova** or the **entire worm** is voided with the stool. *Proglottides* or *segments* can be readily recognized, and may be properly compared with the appearance of flat noodles. Only on superficial examination would confusion with undigested elastic tissue or with the fibrous remains of asparagus be possible. Proglottides are passed singly from time to time, or in case of the Bothriocephalus latus in groups of several in the form of small chains, and facilitate a diagnosis as to the variety of tapeworm. In the fresh state they exhibit movement, inversion, and the like. Proglottides of the Tænia saginata are greater in length than in width, and exhibit upon one side at about the middle a knob-like process—the entrance to the genital canal—and in addition numerous ramifications (from 15 to 20) of the uterus. The latter can be seen with particular distinctness if a proglottis is compressed between two slides. The proglottides of the Tænia solium resemble entirely those of the Tænia saginata, with the exception that the divisions of the uterus are thicker and less numerous (from 5 to 12). The proglottides of the Bothriocephalus latus present an entirely different appearance. In the first place, their width is greater than their length, and, besides, the uterus is arranged in the shape of a roset in the middle (Figs. 34, 35, 36).

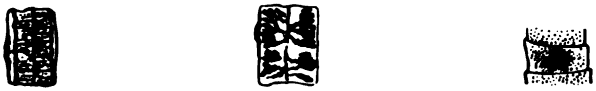

Tapeworm-proglottides; natural size (from personal preparations).

FIG. 34.—Of Tænia saginata. FIG. 35.—Of Tænia solium. FIG. 36.—Of Bothriocephalus latus.

The *ova of tapeworms* are too small to be seen with the unaided eye. They are at times discovered accidentally on microscopic examination of the stools. The differentiation between the ova of bothriocephalus and those of the tæniæ is easy. The eggs of

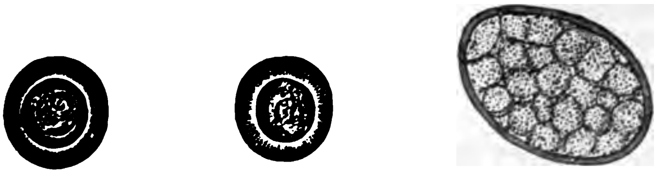

Tapeworm-ova; magnified 370 times (from original preparations).

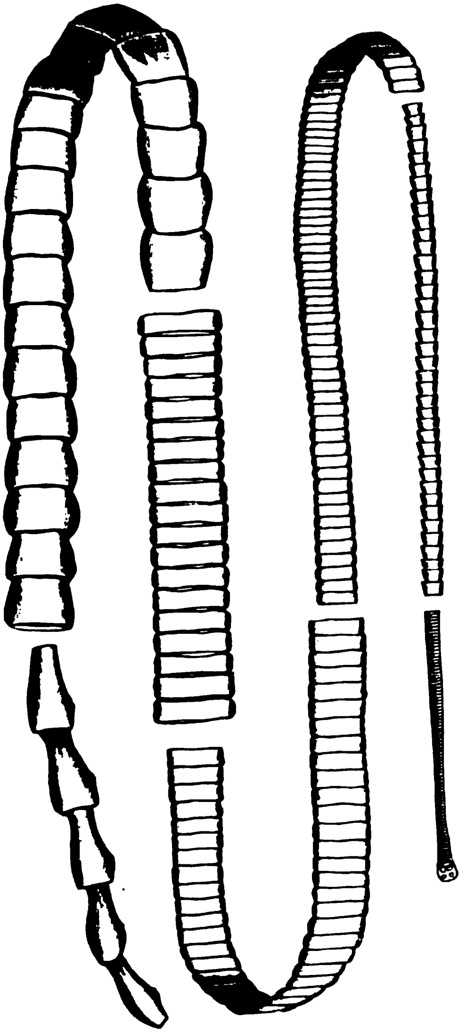
FIG. 37.—Of Tænia saginata. FIG. 38.—Of Tænia solium. FIG. 39.—Of Bothriocephalus latus.

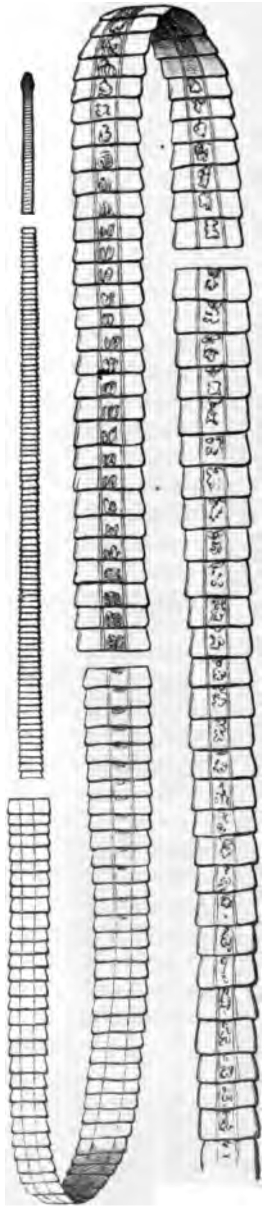
bothriocephalus are oval, present at one extremity a cover, and are filled with a number of spherules. The ova of tæniæ, on the other hand, possess a striated shell, granular contents, and six hooklets in the latter. The differentiation between the ova of Tænia saginata and those of Tænia solium is difficult, as they resemble each other, and differ only in size, those of Tænia solium being smaller (0.032 and 0.036 mm.) than those of Tænia saginata (0.035 and 0.039 mm.) (Figs. 37, 38, 39).

It is rare for *tapeworms* to be expelled entire with the stools. This has been observed in febrile patients, in cases of diarrhea, and during courses of treatment at the springs. In rare instances tapeworms pass from the intestine into the stomach, are in part vomited, and must be extracted with the fingers.

The recognition of the variety of tapeworm naturally can be readily made from the appearance of the individual segments. These become shorter, smaller, and thinner as they approach the head, and the neck is almost filamentous. The head barely attains the size of a pin-head (p. 297, Figs. 40, 41). In the Bothriocephalus latus it has a flat and club-shaped appearance, in the Tænia saginata and the Tænia solium a roundish form. If the head be examined with high powers of the microscope, four suckers are observed; in the Tænia saginata, and in the Tænia solium in addition to four hooklets also an anterior prominence or rostellum, which is surrounded by from 26 to 30 hooklets, and finally in the bothriocephalus two longitudinal deep grooves like suckers (p. 299, Figs. 42, 43, 44). It may be incidentally mentioned that of all tapeworms the Tænia saginata attains the greatest length (up to 8 meters). It may consist of as many as 1300 segments, of which about the six hundredth behind the cervical portion begins to be mature sexually. The Tænia solium attains a length of 3½ meters, and is constituted of as many as 850 segments. The Bothriocephalus latus measures from 5 to 8 meters in length, and is composed of as many as 4000 segments. Tapeworms are found in the *small intestine*, to the mucous membrane of which they adhere by means of their suckers, while the remainder of the body is directed with numerous convolutions toward the anus. In the majority of instances but a single tapeworm is present in the bowel. Should several be present, they are generally of the same variety. In rare instances a number of tapeworm-heads have been observed in an entangled convolution, with their adjacent cervical portions. Injuries of the intestinal mucous membrane are not induced by the application of the suckers of the head of the tapeworm.

Persons harboring tapeworms are often wholly free from subjective symptoms, and become cognizant of the presence of the parasite accidentally through the discovery of the proglottides in the stools. In others a train of symptoms develops; but these are unreliable, and in some instances only a result of the fear of tapeworm. Among suspicious symptoms there have been mentioned irregularity in the action of the bowels, with at times extremely constipated, at other times loose stools, colic, borborygmi, a sense of distortion and twisting in the bowel, excessive secretion of saliva, a disagreeable odor from the mouth, eructation, vomiting, insatiable hunger, but also anorexia, emaciation, and pallor. Earlier physicians reported the occurrence of serious nervous disturbances, such as eclampsia and chorea. Vertigo is not rarely complained of. Inequality of the pupils is often present. The occurrence of Charcot-Neumann crystals in the stools in abundance is suspicious (p. 115, Fig. 20). Recently attention has been directed to profound anemic states in persons harboring Bothriocephalus latus. This *bothriocephalus-anemia* in its





Tapeworms, natural size. (From personal preparations).

Fig. 40.—Tænia saginata.

Fig. 41.—Bothriocephalus latus.

symptomatology completely resembles pernicious anemia, except that retinal hemorrhages and febrile movement are less common with the former. After expulsion of the bothriocephalus the patients often recover with remarkable rapidity. It is assumed that hemogenesis is interfered with through the action of toxic substances (ptomains) generated by the tapeworm, and which develop especially when the parasite has died within the intestinal canal. In cases in which the diagnosis of tapeworm is doubtful a small amount of extract of male fern should be administered and the stools be examined for proglottides and ova:

℞ Extract of filix mas,
Rhizome of filix mas, each, 2.0 (30 grains).—M.
Make 20 pills.
Dose: Take 10 pills morning and evening.

Prognosis.—Tapeworms give rise to an unpleasant but not to a dangerous disease. Only rarely does the host of a tapeworm infect himself with the eggs of proglottides, with the development of measles in other organs, as, for instance, in the subcutaneous connective tissue, the eye, the muscles, or the brain. Caution should be observed in promising success in the treatment of tapeworm, for not rarely the usual remedies prove unsuccessful, and must be repeated. The failure appears, provided efficient medicaments are employed, to depend often upon the fact that the head of the worm is concealed behind a fold of intestinal mucous membrane, and consequently is inaccessible to the action of the drug.

Treatment.—The treatment of tapeworm consists in three parts, namely, the preparatory treatment and the narcotization, or destruction, and expulsion of the worm. All varieties of treatment demand a certain degree of bodily vigor, and they should not be undertaken in convalescents, sufferers from pulmonary tuberculosis and from carcinoma, pregnant women, and those otherwise debilitated. During the period that the teniacide is administered the patient should remain in bed, as otherwise vomiting, vertigo, and syncope may readily take place. The object of the **preparatory treatment** is to empty the intestine as thoroughly as possible, in order that the specific tapeworm-remedy may the better gain access to the parasite, and besides to render conditions unfavorable for the persistence of the worm in the intestine. With this object in view, a mild laxative is given for one or two days, as, for instance, a teaspoonful of licorice-powder in water in the morning, while fruit with small seeds (strawberries, raspberries, gooseberries, currants) should be eaten, and considerable milk, eggs, and meat, but little bread, potatoes, and vegetables, be taken. On the night before the teniacide is administered the patient should take a salad constituted of dried herring, garlic, and onions. The object of the *teniacide* is to narcotize the tapeworm so that it relaxes the grasp of its suckers upon the intestinal mucous membrane, and can be mechanically expelled with the intestinal contents. The most reliable teniacide is freshly prepared **extract of filix mas** given in sufficient dose. We have of late employed with satisfactory results an extract of filix mas enclosed in gelatin-capsules. Gelatin-capsules containing castor-oil may also be given for the mechanical expulsion of the worm. Filix mas may further be prescribed in the following manner:

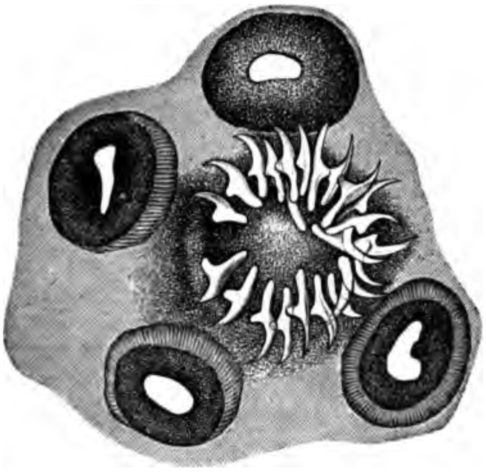
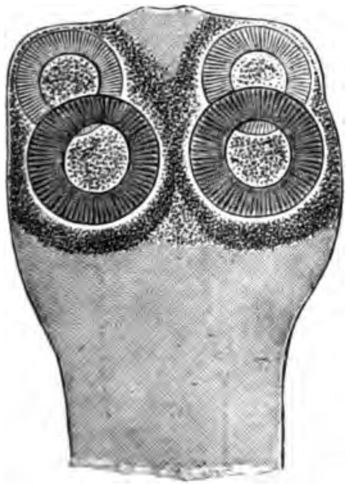
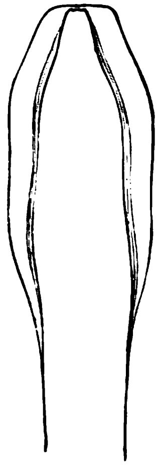


FIG. 42.—Head of Tænia solium.





Tapeworm heads; magnified 60 times. (From personal preparations.)

FIG. 43.—Head of Tænia saginata.

FIG. 44.—Head of Bothriocephalus latus.

℞ Extract of filix mas, 10.0 (2½ drams);
Powdered althea, sufficient to make 30 pills.
Dose: In the morning at 7 and 7.30, when the stomach
is empty, 15 pills.

A tendency to vomit may be counteracted by swallowing black coffee or lemonade.

The number of *tapeworm-remedies* is very large, and of these may be mentioned kousso-flowers (25.0—¾ ounce—to be taken in two parts in the morning in water); koussin (3.0—45 grains—to be taken in the morning in two parts); kamala (15.0—½ ounce—to be taken in the morning in two parts in water); cortex of pomegranate-root (50.0 : 300—1½ ounces : 10 fluidounces—in the morning in two parts); pelletierin (0.3—4½ grains—in two parts); zinc-filings in sirup; and pumpkin-seed.

It is advisable to follow the administration of the teniacide after an interval of two hours by the **expulsive treatment,** unless the bowels have been moved before this time, and the tapeworm at the same time expelled; otherwise the tapeworm might recover, apply itself anew to the intestinal mucous membrane, and neutralize the success of the treatment. The simplest prescription consists of *castor-oil* (50.0—1½ fluidounces) in black coffee. Intestinal infusions of decoctions of the rhizome of filix mas (5.0 : 500—75 grains : 15 fluidounces) may also be advised. Frequently the tapeworm is not expelled entire, but in several parts; or it may happen that a portion is expelled while the remainder is retained within the rectum and protrudes from the anus. In the latter event it is important not to exert traction upon the protruding portion, as the worm may readily tear at the neck; and if this, with the head, remain within the bowel, it may again attach itself to the mucous membrane and attain its former size. After treatment for tapeworm all parts of the parasite passed should be collected and be carefully examined for the head. Should this not be found, the treatment cannot be looked upon as successful. The tapeworm will have acquired its previous size in the course of six months on an average, and will reveal its presence in the bowel by the reappearance of mature proglottides in the stools. The treatment must then be repeated. Some patients are freed from the parasite only after several repetitions of the treatment.

Attention should be given to the **prophylactic measures** against the spread of tapeworm. These will suggest themselves from a consideration of the causative factors, and consist especially in the official inspection of meat and prohibition of the sale of all that is measled, in the avoidance of raw or insufficiently cooked meat, in cleanliness of the stables and preventing the animals from ingesting fecal matter. If the sale of measly meat is permitted, this should be sold separately, from a distinct table, and be indicated as such. The purchaser should be instructed to cook the meat thoroughly. Those engaged in slaughtering should employ special knives for measly meat, so as to avoid the conveyance of measles to healthy meat.

SUCKING WORMS (TREMATODES).

Sucking worms (Distoma crassum, Distoma heterophyes, Distoma hæmatobium) have been found in the intestine only in isolated instances, and have given rise to ulcerative changes.

ROUND WORMS (NEMATHELIMINTHES).

SPOOL-WORM (ASCARIS LUMBRICOIDES).

Symptoms, Anatomic Alterations, Diagnosis, and Prognosis.—The spool-worm in appearance and size resembles the earth-worm. The males are smaller than the females, and at the posterior extremity of the body exhibit an inversion toward the ventral aspect. If spool-worms are passed with the stools, they can be readily recognized. The diagnosis can also be made with certainty if the **ova of spool-worms** can be demonstrated in the stools. These are frequently present in small portions of fecal matter that remain adherent to the anal region. On microscopic examination the egg is found to consist of a granular body, which is surrounded by a thick, bright, wavy, and nodular shell (Fig. 45).

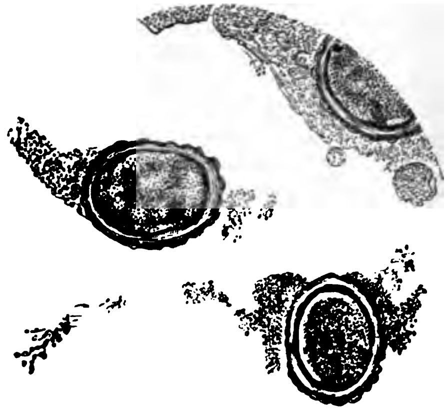


Fig. 45.—Ova of Ascaris lumbricoides: magnified 275 times (personal observation, Zurich clinic).

The **subjective symptoms** are never so distinctive as to render possible a certain diagnosis. The symptoms are in part local, in part reflex (nervous) in nature. Of late it has been properly emphasized that toxic influences may also be operative, because examination of spool-worms has in several instances disclosed the presence of toxic activity (inflammation of the eye, irritation of the skin). The local disturbances include abdominal pain, borborygmi, irregularity of bowel-movement, itching at the anus, anorexia, nausea, vomiting, and offensive odor of the breath. The reflex disturbances include especially certain nervous manifestations, such as vertigo, headache, inequality of the pupils, eclampsia, paralysis, chorea, muscular spasm, and the like. The patient often complains of distressing itching in the nose, and frequently digs the fingers in the nares. He often attracts attention on account of his pallid appearance, the presence of deep rings around the eyes, and progressive emaciation. Spool-worms are not rarely found in the dead body, and one can convince himself that they lodge in the small intestine. At times they occur in such great number that they occupy the entire lumen of the bowel. It is recorded that in one case several thousand parasites were expelled with the stools.

Injury is not inflicted upon the healthy intestinal wall from the presence of spool-worms, and but rarely do they become so entangled as to cause symptoms of obstruction of the bowel. In general, spool-worms are harmless intestinal parasites. Danger arises only from their **migratory tendency.** Spool-worms exhibit a tendency especially to penetrate through narrow orifices. Their entrance into the choledoch duct and their migration upward may readily give rise to obstructive jaundice, which is usually incurable, and at times also to abscess of the liver. Spool-worms have also been found forming the nuclei of gall-stones. Not rarely they invade the stomach, where they occasion a sense of pressure and of pain, and are finally ejected by vomiting, when the pain at once ceases. At times, however, they wander through the stomach and the esophagus during the night into the larynx, lodging above the vocal bands, and causing death by suffocation during sleep. If they advance beyond the chink of the glottis, they may enter deep into the bronchial tubes and excite abscess and gangrene of the lung. At times, also, they enter the nose, the lacrimal duct, the internal ear through the Eustachian tube, and, on perforation of the tympanic membrane, even the external auditory canal, and they must then be removed with the fingers from the nose, the lacrimal caruncles, or the external auditory canal. In cases of perforative peritonitis spool-worms are not rarely found free in the abdominal cavity and in the peritoneal exudate. As a rule, the worms wander into the peritoneal cavity through the opening in the bowel after the perforation has taken place. It should, however, not be considered impossible that in some cases the parasites, with their hard cephalic extremities, may cause perforation of an ulcerative process in the bowel. Not rarely repeated infection with spool-worms occurs if exposure is repeated.

Etiology.—Only those are infected by spool-worms who swallow the ova of the parasites. Such ova have been demonstrated in water, obviously from contamination with fecal matter.

Transmission probably takes place most commonly through **vegetables**, which become infected with the ova of spool-worms in the process of manuring (cattle and pigs often harbor spool-worms) and are not sufficiently washed before being used. Infection may also occur from swallowing with the **food** minute particles of fecal matter that have adhered to the fingers. The greater the want of cleanliness in a household, and the less the care exercised by its individual members, the greater is the danger of transmission. This fact explains the greater frequency of spool-worms in Oriental people, in the insane, and in children.

Treatment.—Spool-worms may be narcotized and expelled with greatest certainty by means of **santonin**, a remedy obtained from the flowers of Artemisia maritima:

℞ Santonin-troches, No. 10.
Dose: 1 troche thrice daily.

The troches officinal in Germany contain 0.025 (⅓ grain) of santonin, but this dose may give rise to toxic manifestations (yellow vision, yellowish discoloration of the skin, the sclera, and the urine, delirium, convulsions).

The **prophylaxis** consists in domestic and personal cleanliness.

In isolated instances the spool-worm of the cat—Ascaris mystax—has been observed in human beings. It is smaller than the Ascaris lumbricoides, and is characterized by the presence of a wing-shaped process at its cephalic extremity. In Greenland the *Ascaris maritima* further occurs.

SEAT-WORM (OXYURIS VERMICULARIS).

Symptoms, Diagnosis, Anatomic Alterations, and Prognosis.—The diagnosis of seat-worms is possible only when the parasites themselves or their ova are present in the stool. The worms are small bodies resembling the maggots of cheese, and presenting a delicate filamentous caudal extremity. The males, which are scarcely half as long as the females, are incurvated at their posterior extremity (Fig. 46). The eggs are oval, and possess granular contents and a bright shell (Fig. 47). The ova can frequently be obtained from remnants of fecal matter at

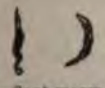


FIG. 46.—Seat-worm (Oxyuris vermicularis): natural size: Female on the left, male on the right. (From personal preparations.)

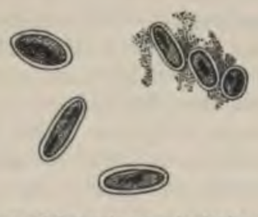


FIG. 47.—Ova of Oxyuris vermicularis; magnified 275 times. (From personal preparations.)

the anus. Not rarely seat-worms are present in the stools in such great number that the fecal matter presents the appearance of a living, swarming mass.. Patients complain in part of local and in part of reflex nervous disturbances.

The **local disturbances** include abdominal pain, rumbling, irregularity in the movement of the bowels, intolerable itching at the anus, anorexia, offensiveness of the breath, nausea, and vomiting. The **nervous symptoms** consist in headache, vertigo, inequality of the pupils, eclampsia, chorea, paralysis, itching of the nose, and the like. The patients often attract attention by their pallor, and their deep and ringed eyes. Seat-worms are found principally in the lowermost portions of the small intestine, and only after fertilization has taken place do the impregnated females migrate into the large intestine, where they may be present in such large number that the mucous membrane acquires a rough and felt-like appearance. Not rarely a number of worms may escape from the rectum during the night, and are found upon the sheets when the bed is prepared in the morning. At times, however, they crawl over the perineum into the vagina, or beneath the prepuce, and give rise to mucopurulent discharges and secretion. The intolerable itching may cause onanism in children. Seat-worms, while not dangerous, may constitute a most obstinate disorder, so that not rarely repeated and thorough courses of treatment may be required before a perfect cure is effected. In the absence of a proper degree of cleanliness repeated infection may take place.

Etiology.—Seat-worms are acquired by the swallowing of ova. Auto-infection may readily take place if small amounts of fecal matter containing the ova remain adherent to the fingers, and are swallowed unconsciously with the food. Children, women, and the insane are the most common hosts of seat-worms. Often infection takes place between sisters and brothers, bed-fellows, and domestic servants.

Treatment.—Efforts should be made to narcotize seat-worms by means of troches of **santonin** (1 troche thrice daily), and besides to remove them mechanically from the intestine by means of **injections into the bowel.** A decoction of finely divided garlic with water, which has stood for twelve hours, and is then strained through linen, may be usefully employed for injection. The infusions should be repeated for several days successively. To prevent migration of the intestinal parasites over the perineum, this should be anointed in the evening with **mercurial ointment.**

WHIP-WORM (TRICHOCEPHALUS DISPAR).

The whip-worm is of no clinical interest, and is usually found in the stools accidentally. It lodges in the cecum, and is characterized by a thin cephalic and a thick caudal extremity, so that it suggests the appearance of a whip. The male is smaller than the female, and its thicker caudal extremity is rolled up toward the dorsal aspect (Fig. 48). The *eggs* of the parasite are oval granular bodies, with a bright sheath, presenting knob-like swellings at either pole (Fig. 49). Infection probably takes place through the ingestion of ova with insufficiently cleansed vegetables. In the case of a child it took place through the eating of garden-earth, in which the ova of trichocephalus were demonstrated.[1]

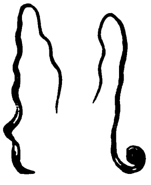


FIG. 48.—Whip-worm (Trichocephalus dispar); male to the right, female to the left; natural size (from personal preparations).

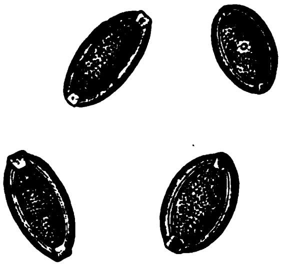


FIG. 49.—Ova of whip-worm (Trichocephalus dispar) magnified 275 times (from personal preparations).

TRICHINA SPIRALIS.[2]

Etiology.—As a rule, human beings are attacked by *trichiniasis* (*trichinosis*) in consequence of the ingestion of **pork or ham containing trichinæ**. Only exceptionally does infection take place through other animals, as, for instance, the wild boar. If trichinous meat is eaten, muscle-fibers and trichina-capsules are dissolved in the process of gastric digestion, and the muscle-trichina of the meat are set free. These enter the small intestine, develop within two and a half days into *intestinal trichinæ*, engage into sexual relations, and in the course of a week the females reproduce numerous young. While the parent ova die in the course of from five to eight weeks, and are expelled from the intestine with the stools, the young brood wanders from the intestine, and the parasites find their way into the muscles. Here they penetrate the muscle-fibers and are subsequently surrounded by a capsule. They have thus become *muscle-trichinæ* in the human being. Trichiniasis includes the period of time included between the ingestion of trichinous meat and the final transformation of the brood of trichinæ into the muscle-trichinæ in the human being.

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[1] Several cases of extreme anemia due to the Trichocephalus dispar have been reported.—A. A. E.

[2] Trichinella spiralis.—A. A. E.

As a rule, trichiniasis occurs in *epidemic* form, and this is readily explicable from the fact that a trichinous pig is used as food by many persons at the same time or within a short period. In many instances epidemics of trichiniasis, in which several hundred persons were attacked, have originated from a butcher-shop in which a trichinous pig had been offered for sale. In other instances smaller circles of relatives and friends were attacked who had partaken together of a given meal or had received some part of the pig as a gift. Epidemics in barracks have also been observed. Epidemics of trichiniasis are more common in certain *regions* than in others. The Harz region has a bad reputation in this respect, whereas Switzerland, France, and England have heretofore remained entirely free. This fact is dependent upon local custom, as, for instance, in the Harz upon the practice of eating raw meat. Besides, trichiniasis occurs with varying frequency among swine in different regions. It occurs with especial frequency in America, and attention therefore has properly been called to the fact that the use of American ham and bacon may be attended with serious dangers.[1]

The opportunity for swine to become trichinous is the more favorable the greater the *want of care and of cleanliness in the pens* in which they are kept. If the animals are not prevented from eating one another's feces, it may readily happen that if accidentally a trichinous pig is introduced into the herd, this animal may infect the remainder through its trichinous stools. A still greater source of danger of infection is constituted by **trichinous rats**, as these animals are frequent occupants of pig-pens and are readily eaten by the pigs. The rats in turn acquire their trichinæ from the ingestion of the trichinous fecal matter of the pigs. As rats frequently migrate from one pen to another, the danger of their being the means of suddenly conveying trichiniasis into herds of healthy swine must be obvious. Whether rats or pigs were originally the hosts of trichinæ is still a disputed question. The *mode of fattening pigs* has an important bearing upon the development of trichinæ in these animals. In America particularly it is customary to feed pigs at rendering establishments with the meat of dead animals; and as care is not taken to determine whether such meat is free from trichinæ, trichiniasis is often conveyed to the hogs in this way.[2]

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[1] While trichiniasis is common in hogs in the United States, it is comparatively rare in man, because pork is usually well cured or cooked.—A. A. E.

[2] This statement requires modification. All meat for interstate consumption and for export is submitted to governmental inspection, and the practice of feeding the offal of slaughtered animals to hogs would be interdicted, even if the land about abattoirs were not too valuable to forbid its use for growing hogs. such a practice is followed at some country slaughter-houses.—

All animals that partake of trichinous meat may be attacked by trichinæ. The disease has been intentionally produced even in salamanders. Cats are not rarely trichinous from having eaten trichinous mice or rats. Trichinæ have been found also in the muscles of wild animals (badger, polecat, fox), probably from the ingestion of trichinous mice or rats.

Anatomic Alterations.—If impregnation of ingested trichinæ has taken place in the intestine of a human being, the birth of a new brood of trichinæ begins on the seventh day. The young animals quickly leave the intestine, to migrate into the muscles. The paths that they follow in their wanderings are known only in part; at any rate, the lymphatics and also perhaps the blood-vessels are followed. Trichinæ are found with great constancy in certain groups of muscles. Among these are especially the diaphragm, the intercostal muscles, those of the vocal bands, the neck, the eyes, and those of mastication. On the other hand, the myocardium is almost unexceptionally exempt from invasion. In the muscles of the extremities the trichinæ are found in greatest number in the vicinity of the tendons. In the muscles the young trichinæ induce especially parenchymatous myositis. In those situations in which trichinæ are contained within a muscular fiber this swells, and its protoplasm acquires a glistening appearance free from transverse striation. At the same time proliferation of the sarcolemma-nuclei takes place. Vacuolation, waxy degeneration, and granular disintegration of the muscle-fibers occur. Further, inflammatory hyperplasia and cell-multiplication take place in the interstitial connective tissue. After a time the trichinæ, at first extended, become rolled up spirally, and surrounded by a lemon-shaped *capsule* (Fig. 50). Later, calcification of the capsule takes place. Within their capsule trichinæ have at times persisted throughout a life of several decades. Occasionally, however, the process of calcification involves the trichinæ themselves, and they may as a result become disintegrated into several calcified segments. Whether trichinæ in muscles possess vitality or not may be recognized from the fact that on microscopic examination of teased preparations of trichinous muscle living trichinæ are motile if the preparation be placed upon a warm stage. Not rarely several trichinæ may be found within a single muscle-fiber. Two trichinæ, rarely more, may also be enclosed within a common capsule. Trichinæ may be discernible in the muscles even with the unaided eye, for the capsules appear as small, light-yellow points. At times the muscles are fairly crowded with such yellow points. Naturally, confirmation with the microscope should be made, and for this purpose a magnification of 50 is quite sufficient. I have repeatedly found at autopsy old muscle-trichinæ accidentally and as a secondary phenomenon in human beings. When individuals die as the result of trichiniasis they frequently present **edema of the eyelids and of the extremities.** Often the **muscles of mastication** are unusually well defined beneath the skin of the face in consequence of marked contraction, and the **elbow-joints** are greatly flexed. The **face** appears deeply sunken. The voluntary **muscles** are thickened and turbid, and exhibit here and there extravasations of blood. Such extravasations may be present also in the myocardium. The **spleen** presents the appearance common to infectious diseases, and is large and soft in consistency. The **liver** and the **kidneys** appear swollen and cloudy, and also fatty. The **mucous membrane of the stomach,** and especially of the **small intestine,** exhibits inflammatory swelling and often extravasations of blood. It is important to obtain the intestinal contents, to dilute them with water, and to examine them for intestinal trichinæ and their offspring. The trichinæ can just be recognized as small, grayish-white filaments, the female being from 3 to 4 and the male on the average 1.5 mm. long.

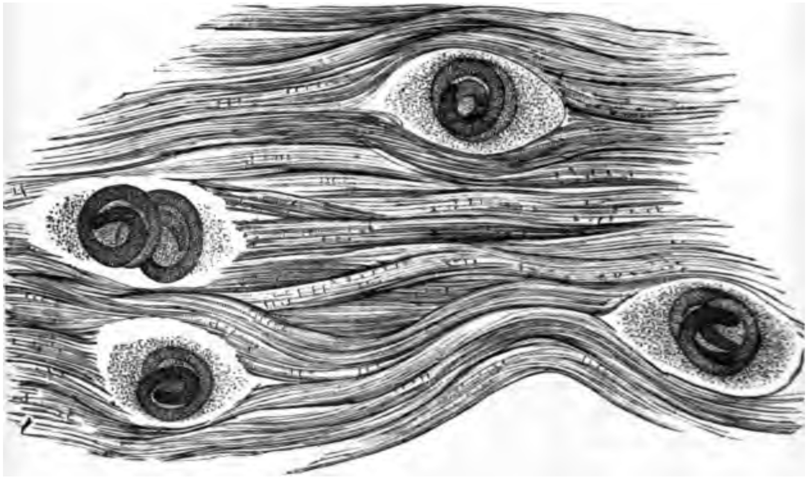

FIG. 50.—Muscle-trichinæ with capsules, from the diaphragm; magnified 60 times (from personal preparations).

Symptoms.—Persons who have eaten trichinous meat remain well, as a rule, for a week. It is but rarely complained that immediately after the ingestion of trichinous food malaise, nausea, vomiting, and diarrhea have set in. Generally the symptoms named appear after the lapse of a week, following the birth of the young trichinæ in the intestine. The diarrhea may at times be so excessive that the clinical picture is suggestive of Asiatic cholera, and death may result from exhaustion. Soon **alterations in the muscles** are superadded. The patient complains of **severe pain** in the muscles and in the extremities, which already appear sensitive to the slightest touch, and feel tense and doughy. In consequence of involvement of the muscles of the vocal bands hoarseness results; the eyes are painful on movement, and this becomes difficult; breathing becomes embarrassed and painful; and also in the extremities stiffness and intolerable pain appear on any attempt at movement. In many instances **muscular contractures** and **fixed positions of the extremities** develop. Flexion at an acute angle takes place at the elbow-joints, and also in the hip-joints and the knee-joints; the masseters are contracted with board-like hardness, and lock-jaw or trismus results. I have also observed stiffness in the muscles of the neck and the back, so that distinct opisthotonos developed, the vertebral column being greatly curved forward, and the patient resting only with the occiput and the sacrum upon the bed. To the distressing pains in the muscles there are thus superadded stiffness and immobility of the entire body, and the patient requires assistance for all movement. The occurrence of **edema of the skin** is, further, noteworthy. This is noticeable especially in the eyelids, but also in the extremities. Its cause is not definitely known. **Symptoms of general infection** further play an important part in the clinical picture of trichiniasis. These may perhaps be dependent upon the activity of toxic substances excreted by the trichinæ, but perhaps they may be due to the absorption of products of abnormal metabolism developed in consequence of the extensive inflammation of the muscles. Trichiniasis is, as a rule, attended with **fever**, and the temperature may reach 39° C. (102.2° F.), 40° C. (104° F.), and even more. The temperature does not pursue a definite course, and periods of continued and remittent fever alternate with each other. Often the fever extends over several weeks. In correspondence with the elevation of bodily temperature thirst is increased, while the appetite suffers. Trichinous patients almost constantly suffer from copious **sweating**. The face and the trunk are usually covered with large drops of sweat, and often the skin in the portions of the body covered by clothing is raised by stagnant sweat into innumerable, small, transparent, dewdrop-like vesicles—sudamina or miliaria. Frequently the patient complains of persistent **sleeplessness—agrypnia**. The abdomen is often greatly distended. The **spleen** can be demonstrated to be enlarged by percussion, and frequently also by palpation. The coated tongue and the persistent diarrhea, in conjunction with a portion of the symptoms described, often excite suspicion of typhoid fever, a suspicion that can readily be removed by showing that the blood-serum of the trichinous patient fails to cause clumping and loss of motility of typhoid-bacilli. The **urine** is usually passed in small amounts, although later polyuria may develop. **Albuminuria** is an uncommon symptom, and is usually of febrile

[Trichiniasis is attended with a marked increase in the number of white blood-corpuscles, and it is characteristic that the augmentation involves particularly the eosinophile cells.—A. A. E.]

Trichiniasis generally pursues a *subacute* or a *chronic course.* Frequently the strength grows progressively less, pronounced marantic edema sets in, and the patients die from exhaustion after weeks of misery. Occasionally death takes place from pneumonia amid manifestations of progressive respiratory disturbance. Among the *complications septic manifestations* are especially to be named, as, for instance, furunculosis and suppurative parotiditis. Should recovery from the disease take place, muscular and articular pains and stiffness, at times muscular induration and great irritability of the abdomen and the intestine, frequently persist for a long time.

Diagnosis.—The recognition of trichiniasis is easy and certain if small bits of muscle are excised and examined microscopically for trichinæ. The biceps of the arm is the muscle best suited for this purpose, as it contains trichinæ with great constancy. It may also be important to examine the stools for trichinæ. Inquiry should further be made to determine whether any of the meat partaken of can still be secured, and this also should be examined for trichinæ. It is always suspicious when a number of persons who have taken part in the same meal, or have obtained their supply of meat from the same butcher-shop, are attacked with the symptoms described. Formerly trichiniasis was most commonly mistaken for typhoid fever, or for obstinate and serious varieties of muscular or articular rheumatism. A similar clinical picture is presented by *acute polymyositis*, but this disorder occurs independently, apart from the ingestion of meat, and only in isolated instances.

Prognosis.—The prognosis of trichiniasis is exceedingly grave, as no remedy is known that is capable of preventing the migration of young trichinæ from the intestine into the muscles and of destroying trichinæ present in the muscles. In some epidemics of trichiniasis death occurred in one-third of those attacked.[1]

Treatment.—If a number of persons have partaken of trichinous meat from the same animal, experience has shown that the severity of the disease may be most variable. This will depend, in the first place, upon the fact whether one person has eaten more and another less of the meat, whether alcohol was used at the meal, and whether this was possibly followed by vomiting and diarrhea. Under favorable conditions trichiniasis may not develop in one or another of those who have partaken of infected meat. It is rare for persons to observe soon after ingestion that the meat partaken of was trichinous. Under such circumstances the contents of the stomach should be at once removed

[1] The mortality among large numbers of cases is about 5 per cent.—A. A. E.

by **irrigation,** and the intestinal contents evacuated as rapidly as possible by means of **castor-oil** (30.0—1 fluidounce). In addition the administration of **glycerin** internally has also been recommended (15 c.c.—1 tablespoonful—every hour), as trichinæ die rapidly in this substance. When trichiniasis has fully developed, only **symptomatic treatment** is available. Severe pains can be relieved by *hot baths* (from 28° R.—95° F.—to 30° R.—99.5° F.), and *subcutaneous injections of morphin.* The *diet* should be light yet nutritious. Profuse sweating is best relieved by means of *atropin sulphate* (0.0005—$\frac{1}{125}$ grain—once or twice daily). Within the first eight days an effort should be made to expel any trichinæ possibly present in the intestine, as, for instance, by:

℞ Santonin, 0.05 (¾ grain);
Mercurous chlorid,
Jalap,
Sugar, each, 0.5 (7½ grains).—M.
Make 5 such powders.
Dose: 1 powder twice daily.

Great importance is to be attached to the **prophylaxis.** As has been indicated in the consideration of the etiology, this includes cleanliness in the pig-pens, caution in the feeding of hogs with the meat of slaughtered animals, thorough meat-inspection, the destruction of trichinous meat, and the avoidance of uncooked or insufficiently cooked pork. Pickling and smoking of pork are not capable of removing with certainty all danger of infection.

ANKYLOSTOMUM DUODENALE.[1]

Etiology.—Until within a few years it was believed that *ankylostomiasis* occurred only in the Orient and in Italy. In the course of construction of the St. Gotthard tunnel it developed that the disease was introduced into Switzerland by Italian laborers. Foci of the disease were then observed in certain mines of France, Sardinia, Belgium, and Hungary, and in recent years the disorder has been encountered in brick-yards at Bonn, Cologne, Würzburg, and Berlin. As the most prominent symptoms consist in progressive anemia, the disease has been given the designations *tunnel-anemia, miners' anemia, brickmakers' anemia,* in accordance with the conditions under which it occurs. In Egypt, where the disease has been known for some time, it has been named *tropical chlorosis.* It has been repeatedly demonstrated with certainty that the disease is spread by foreign wandering laborers, who come from localities in which ankylostomum occurs, and where they have themselves contracted ankylostomiasis. The danger of infection becomes the greater the more intimately the sick and the well live together and the less the degree of care observed in the disposition of the fecal discharges in secure and closed receptacles, in order to prevent their entrance into the water used for drinking and other domestic purposes. Infection usually takes place through water polluted with fecal matter containing ankylostomum-ova, or through particles of earth containing larvæ of ankylostomum and adhering to the fingers, and which may be readily swallowed when food is ingested.

Anatomic Alterations.—The Ankylostomum duodenale is a small cylindrical worm (Fig. 51); the females, of which there may be more than twice as many as of the males, are longer (up to 18 mm.) than the males (from 6 to 10 mm.). The number of the parasites in the intestine may vary between 15 and 3000.

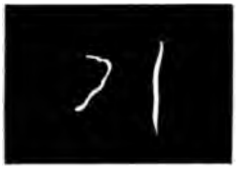

FIG. 51.—Ankylostomum duodenale: the male to the left, the female to the right (natural size).

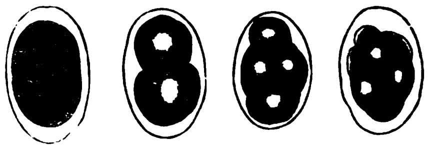

FIG. 52.—Ova of ankylostomum.

The eggs of ankylostomum are found in the stools in almost uniform distribution, and they may be readily recognized on microscopic examination. They are of oval shape, and possess a bright shell and granular contents, which are usually in a state of fission (Fig. 52). Ankylostoma are found much more constantly in the *jejunum* and the *ileum* than in the duodenum. They attach themselves by suction to the intestinal mucous membrane by means of their mouth, which is armed with six chitinous teeth, and thus extract blood from their host. The females appear to be especially bloodthirsty, and they suck much more blood than is necessary for their existence. They therefore present a brownish appearance, dependent upon the blood collected within their body. On post-mortem examination ankylostoma are found free in the intestinal contents or firmly attached to the *intestinal mucous membrane.* In the latter event they are often engaged in rolling movements. The intestinal mucous membrane exhibits in places recent as well as older extravasations of blood and hemorrhagic suffusions that correspond to the points of attachment of the parasites. In addition, all of the viscera are conspicuous for their exceeding **anemia** and **pallor.** In consequence hemorrhage has taken place in various situations. These are found with especial frequency upon the inner aspect of the cerebral dura mater, where they are often converted into pachymeningitic membrane. **Anemic fatty degeneration** also generally develops, particularly in the myocardium, the cells of the liver, and the epithelial cells of the convoluted uriniferous tubules. The **bone-marrow** appears deficient in fat, and at times contains nucleated red blood-corpuscles in unusual number.

Symptoms.—The symptoms of ankylostomiasis consist in gradually progressive **anemia** and its consequences, and are dependent upon abstraction of blood by the parasites. Whether, in addition, toxic influences are also operative has not yet been demonstrated. On the average anemic manifestations appear within five or six weeks after infection has taken place. The patients acquire a **pallid appearance,** which may attain such a marked degree as to create a cadaveric aspect. *Examination of the blood* discloses the fact that the number of red corpuscles may be diminished to one-fifth of the normal, and that also the hemoglobin-percentage is diminished correspondingly. At the same time the red blood-corpuscles are characterized by their variability in shape—poikilocytosis. They also vary greatly in size, and assume the appearance of a pear, a club, a figure of eight, and the like. At times nucleated red blood-corpuscles also are found. The greater the degree of anemia the more markedly do the **functions of the body** become impaired. The patient tires readily from work, suffers from dyspnea and palpitation of the heart, and is attacked by sweats. Finally, the weakness becomes so excessive that the patients are compelled to remain in bed constantly because vertigo, tinnitus aurium, confusion of vision, and syncope may occur in the erect posture,—all signs of increasing cerebral anemia on assumption of the erect posture. The **heart** appears enlarged toward the right (anemic dilatation of the less resistant, because thin-walled, right ventricle), and over it anemic systolic murmurs may be audible. Over the bulb of the internal jugular vein a loud venous hum or "nun's murmur," sometimes palpable as a thrill, may be detected. A cardiac-systolic arterial sound is audible over the large peripheral arteries (brachial, radial). Not rarely **cutaneous edema** develops, because the blood-vessels, whose nutrition has been impaired, are excessively permeable to the blood-plasma. Accumulations of fluid (transudates) may take place also in the serous cavities (pleura, pericardium, peritoneum, meningeal spaces). Nutritive disturbances in the blood-vessels give rise also to the occurrence of **hemorrhages,** which may take place into the external integument, the mucous membranes, and in isolated instances also the retina, and under circumstances may increase the anemia to a considerable degree.

The **appetite** is, as a rule, impaired, while **thirst** is greatly increased. At times there may be paradoxic appetite—**allotriophagy**—for inedible and indigestible substances, as, for instance, earth (geophagy). The stools may then contain also sandy and earthy constituents. At times they contain blood. The occurrence of ankylostomum-ova is important, while ankylostoma themselves are but rarely encountered. Usually the stools contain a large number of Charcot-Neumann crystals (p. 115, Fig. 20). The bowels are at times constipated, at times loose. Frequently there is complaint of a sense of pressure, of fulness, and of pain in the abdomen, which is distended from meteorism. The **urine** is usually passed in large amounts, and contains a good deal of indican. The **temperature of the body** is often subnormal, although febrile movement may occur for periods of greater or lesser duration (anemic fever). Should no attempt be made to expel the parasites, or should this be successful only after the anemia has become excessive, the patients may die like the victims of hemorrhage. The bodily weakness becomes excessive and consciousness obscured, and finally death takes place. The disorder is usually chronic in course. Improvements and exacerbations take place, the former after expulsive treatment, and the latter when the results of treatment are not entirely successful.

Diagnosis.—Ankylostomum-anemia resembles perfectly **progressive pernicious anemia,** and a differentiation can be made only from the fact that in cases of pernicious anemia the stools are free from ankylostoma and ankylostoma-ova. The latter circumstance likewise distinguishes ankylostomiasis from all other anemic states—in old persons, for instance, from carcinomatous cachexia. A positive opinion as to the absence of parasites and ova from the stools will naturally be possible only if the intestinal discharges remain unchanged after the administration of extract of filix mas.

Prognosis.—The prognosis of ankylostomiasis is serious, because some patients are already in such an anemic and exhausted state when they first come under medical observation that death soon occurs in spite of expulsive treatment. The likelihood of recovery is further not rarely lessened by the fact that occasionally the patient declines thorough treatment because he does not have time to give up his work and submit to proper treatment in a hospital for a sufficiently long period. In cases in which the instructions of the physician will be followed and professional aid is not sought too late, recovery can be promised.

Treatment.—**Extract of filix mas** has proved the most certain remedy for ankylostomiasis, but it must be given in large doses (from 10.0—2½ fluidrams—to 20.0—5 fluidrams). The preparation for tapeworm mentioned on p. 300 may also be employed. Frequently ankylostoma remain in the intestine, and the males especially appear, by reason of their small size, to be able to conceal themselves readily behind folds of the intestinal mucous membrane, and thus to escape the action of the remedy. For this reason the stools must again be examined for ankylostoma-ova a short time after the treatment, and if eggs be found present the expulsive treatment should be repeated. The anemic conditions induced as a result of ankylostomiasis should be corrected by means of nutritious food, arsenic, and mild preparations of iron.

The **prophylaxis** is important. This consists in excluding the carrier of ankylostoma from work-places, enjoining the laborers to eat only with carefully cleansed hands, and exercising care to secure perfect closure of water-closets and of water-conduits.

ANGUILLULA INTESTINALIS AND ANGUILLULA STERCORALIS.[1]

Anguillula intestinalis and Anguillula stercoralis have been found in the stools in some cases of tropical diarrhea, especially *Cochin-China diarrhea*, although they have been found also in the laborers on the St. Gotthard tunnel in conjunction with the Ankylostomum duodenale. In all probability these are not two different varieties of worm, but the Anguillula stercoralis represents an earlier stage in the development of the Anguillula intestinalis. The latter occurs especially in the small intestine, and the Anguillula stercoralis throughout the entire large intestine. On microscopic examination the Anguillula intestinalis is found to be a circular worm, with a conical caudal extremity, within whose interior the digestive tube with two enlargements can be seen (Fig. 53). *Thymol* (1.0—15 grains—every hour) especially has been recommended for the expulsion of the parasites.

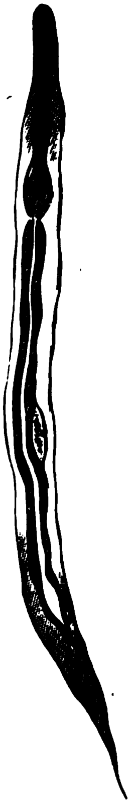

FIG. 53.—Anguillula intestinalis; magnified 100 times (after Seifert).

The larvæ of flies have in a number of instances been found in the stools, having been swallowed with food. The patients have complained of pain in the abdomen, and have at times suffered from obstinate diarrhea.

VI. DISEASES OF THE LIVER.

JAUNDICE (ICTERUS).

Etiology.—Jaundice is an exceedingly common symptom of disease of the liver and biliary passages. Among diseases of the liver fatty liver and amyloid liver alone are unattended with jaundice in the absence of complications. Jaundice occurs most commonly in consequence of **obstruction of the biliary passages,** whether this involves the larger ducts or those within the liver. The cases are much less common in which in the presence of **thrombosis of the portal vein** the blood-pressure in the intrahepatic branches of this vessel becomes so slow that, contrary to the rule, the bile secreted by the liver-cells passes over, not into the biliary capillaries, but into the branches of the portal vein. In rare instances the bile stagnates in the intrahepatic biliary passages because the **movements of the right half of the diaphragm** are interfered with, and, as a result, the inspiratory pressure of the diaphragm upon the liver and the mechanical propulsion of the secreted bile toward the intestine are removed. For this reason jaundice is sometimes observed in association with right-sided diaphragmatic pleurisy. Occasionally the biliary passages become so greatly distended with bile that they are incapable of conveying all of this secretion to the intestine, and a portion thereof passes over into the blood-vessels and lymphatics. This condition has been described as jaundice due to *polycholia* or *pleiochromia.* Such conditions arise especially after the ingestion of certain poisons that cause dissolution of the red blood-corpuscles. The hemoglobin set free in the blood-current is acted upon by the liver-cells, and converted into biliary coloring-matter, and pleiochromia results.

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[1] Strongyloides intestinalis.

Under all of the conditions thus far mentioned the jaundice results from *mechanical, hypostatic,* or *resorptive* influences. In recent years attention has been directed to another variety of jaundice, which has been designated *diffusive* or *acatectic.* Under normal conditions it is the function of the liver-cells not only to produce bile from blood coloring-matter, but also to convey the bile into the biliary passages. Under morbid conditions the latter property may be lost, so that the bile produced by the liver-cells is not discharged into the biliary passages, but into the lymphatics and the blood-vessels. It has been assumed that such a diffusion-icterus may develop in consequence of the action of poisons, ptomains, and toxins. The theory of diffusion-icterus, however, still requires more convincing evidence, and it appears premature to attempt to explain almost all varieties of icterus as due to diffusion.

Among all of the varieties of jaundice thus far mentioned that is the most common which develops in consequence of obstruction of the biliary passages, and which has long been known as *hepatogenous, mechanical, resorptive,* or *hypostatic icterus. Obstruction of the larger biliary passages* occurs with especial frequency in connection with gastro-intestinal catarrh, either from constriction or occlusion of the mouth of the choledoch duct in consequence of swelling of the duodenal mucous membrane, or from the entrance of a plug of mucus from the inflamed mucous membrane into the choledoch duct, or, finally, the mucous membrane of the choledoch duct has become involved in the inflammation of the intestinal mucous membrane, and the inflammatory swelling of the mucous membrane of the duct has given rise to obstruction of its lumen. The jaundice resulting from gastro-intestinal catarrh is designated *catarrhal* or *gastroduodenal.* Not rarely the biliary passages are obstructed by *foreign bodies.* Most commonly these consist of *gall-stones,* and only exceptionally of distomata or spool-worms, which have migrated from the duodenum into the choledoch duct, or fruit-kernels and foreign bodies from the food. *Cicatricial* and *carcinomatous obstructions of the biliary passages* are not common. The former usually result from gall-stones that have injured the mucous membrane of the biliary passages in their progress. At times the larger biliary passages are obstructed by *pressure from without.* The most common causes of this condition are tumors of the stomach, the intestine, the pancreas, the kidneys, wandering kidney, peritoneal exudates, bands, aneurysms of the hepatic artery, enlargement of the uterus or ovaries. Enlargement of the portal lymphatic glands may also be mentioned as a cause of obstruction of the biliary passages. Obstruction of the intrahepatic biliary passages and the resulting jaundice are, as has been mentioned, frequent symptoms of the most varied *diseases of the liver.*

It was formerly assumed that jaundice might be *paralytic* or *spastic,* the former depending upon paralysis, the latter upon spasm of the biliary passages, and upon the secondary biliary stasis. This view is unfounded and improbable.

Opinions are divided as to the causes of *toxic jaundice,* and probably various disturbances are operative in this connection. Thus, in cases of jaundice due to phosphorus-poisoning catarrh of the smallest biliary passages in the liver has been found. Other poisons (ether, chloroform, hydrogen arsenid) are destructive of the red blood-corpuscles, and give rise to jaundice in consequence of polycholia or pleiochromia. Diffusion-icterus appears to develop especially in consequence of the action of *bacterial poisons—toxins.* Jaundice is observed not at all rarely in the course of certain infectious diseases (typhoid fever, septicopyemia, diphtheria, relapsing fever, pneumonia, etc.), although other accidental influences, as, for instance, gastro-intestinal catarrh, may give rise to jaundice under such conditions, and this possibility must be carefully considered in the individual case.

Symptoms.—The most conspicuous symptom of jaundice is the **yellowish discoloration of the skin, the urine, and the mucous membranes.** This results from the presence of biliary coloring-matter in the blood. In part the blood takes up the biliary coloring-matter directly from the liver, but in the presence of biliary stasis the lymphatic vessels play by far the more important part in the absorption of bile, which in turn they convey to the blood. *Cutaneous jaundice* is characterized by a yellowish discoloration of the skin, the intensity of which varies with the amount of biliary coloring-matter circulating in the blood from a light sulphur-yellow to a coppery-brown or yellowish-gray. In the latter event the designation *melasicterus* has been employed. The yellowish discoloration becomes first apparent in places where the skin is thin and well supplied with vessels, therefore earliest in the face, and here especially in the temporal region, upon the forehead, on the nasolabial folds, and upon the chin. Upon the forearms and the legs the yellowish discoloration of the skin is often less pronounced because the epidermis is thick and in laborers is brown from exposure to the rays of the sun. While at the outset cutaneous jaundice is dependent solely upon the yellowish discoloration of the blood-plasma by the biliary coloring-matter, subsequently the cells of the epidermis become saturated with biliary coloring-matter, which crystallizes in the form of brownish granules in the epithelial cells. This fact explains the darker color of the skin when jaundice has existed for some time, as well as the experience that cutaneous jaundice may persist much longer than the remaining manifestations of jaundice, for it disappears only when the epidermic cells discolored by the biliary coloring-matter are desquamated.

Icterus of the mucous membranes is most readily recognized in the conjunctivæ. The scleræ appear more or less deeply yellow, instead of white. Usually this icterus of the conjunctivæ is an early manifestation of jaundice, and in mild cases discoloration of the skin and of the urine may be absent, and only that of the conjunctiva be present. Upon other mucous membranes (lips, mouth, and pharynx, labia and vagina) the jaundice is concealed by the red color of the mucous membrane; but if pressure be exerted and the blood-vessels are thus emptied the icteric discoloration of the mucous membrane becomes distinct. In the buccal cavity when the mouth is widely opened, two bands of yellowish mucous membrane, increasing in size posteriorly in the course of the middle line of the hard palate, are visible. *Icteric urine* is discolored brown, blackish brown, at times greenish brown, on agitation yields a yellowish foam, stains white paper, linen, and other materials yellow, and on standing retains its foam for an unusually long time. The presence of biliary coloring-matter in the urine may be best demonstrated by means of Gmelin's or Maréchal's test. The presence of biliary acids can generally be demonstrated also by means of Pettenkofer's test. Tube-casts occur constantly in the urine, most frequently hyaline, and often stained yellow by biliary coloring-matter. Slight albuminuria also is not uncommon. Cylindruria and albuminuria result from the interference by the biliary acids circulating in the blood with the activity of the epithelial cells in the convoluted urinary tubules.

Gmelin's test for biliary coloring-matter is performed in the following manner: A test-tube is filled one-third with nitric acid containing some nitrous acid, and from another test-tube filled with urine this fluid is permitted to flow slowly upon the nitric acid, most conveniently by holding the two tubes obliquely in relation with each other at their mouths, so that the urine overlies the acid and forms a layer upon it. If the urine contain bilirubin, a play of colors takes place at the line of contact with the nitric acid, and from below upward rings of yellow, red, blue, and green will be seen to develop successively. The green ring alone is distinctive of biliary coloring-matter, as a brownish ring occurs also when the urine contains considerable indican. If the urine contain a small amount of bilirubin, it is advisable to pass the urine through filter-paper, in order to concentrate the bilirubin thereon, to spread the filter upon a porcelain dish, and then to apply a glass rod that has been dipped in impure nitric acid. At the point of contact the ring of color mentioned will appear, the red being innermost and the green outermost.

Maréchal's test for biliary coloring-matter requires even less skill than Gmelin's. To urine with which a test-tube has been half filled several drops of tincture of iodin are added, or, still better, a solution of iodin and potassium iodid—so-called Lugol's solution, which consists of pure iodin, 0.5 (7½ grains); potassium iodid, 5.0 (75 grains); and water, 200 (6½ fluidounces). The presence of bilirubin in the urine is disclosed by the development of a grass-green or an emerald-green color. Gmelin's and Maréchal's tests are not always equally distinct with the same urine, and it is possible that only one or the other may yield a positive reaction. In my experience Maréchal's test fails the less commonly.

Pettenkofer's test for biliary acids depends upon the fact that these acids when gently heated with pure sulphuric acid and sugar give rise to a carmine color. As, however, it would be difficult for the practising physician to obtain the biliary acids in pure form from the urine, Strassburger has proposed the following procedure, which in my experience has proved wholly practicable: A bit of sugar is dissolved in the urine, and then a piece of white filter-paper is dipped in the solution and permitted to dry. The filter-paper is then touched with a glass rod carrying a drop of pure, concentrated sulphuric acid. If the urine contain biliary acids, a carmine-red color appears at the point of contact, while, otherwise, a blackish-brown color at once appears in consequence of the action of the sulphuric acid.

A number of other symptoms appear in the clinical picture of jaundice, but they are rather of subordinate importance in comparison with the yellowish discoloration of the skin, the mucous membranes, and the urine. **Alterations in the frequency of bowel-movement** and in the **stools** are among the most common. If jaundice be the result of biliary stasis, the patient usually suffers from constipation, for if no bile enters the bowel the stimulating effect of the biliary acids upon the intestinal musculature will be wanting. At the same time the stools consist of large nodular masses. Usually they give off an offensive, putrid odor, because the antiseptic influence of the bile upon the intestinal contents has been withdrawn. The color of the stools is also striking, and, accordingly as the flow of bile to the intestine is restricted or abolished, it varies between that of clay and that of ash. At times the stools present almost a silvery luster. This is due to the large amount of fat present, and this in turn is dependent upon the fact that the biliary acids in the intestine aid in the absorption of the fat in the food, so that in the presence of biliary stasis and deficiency of bile in the intestine the absorption of fat is diminished. If the stools be stirred in water, drops of fat collect upon the surface on standing, and if these be examined microscopically innumerable delicate needles of sodic or magnesic soaps will be found. The stools retain their bilious color in the presence of jaundice if the obstruction to the flow of bile through the intestine is not complete, or if the icterus be of pleiochromic or diffusive origin. Jaundiced persons are often annoyed by distressing **itching of the skin—cutaneous pruritus**—the existence of which may disclose itself to the eye by the presence of scratch-marks and crusts upon the skin. The itching frequently occurs at night, and deprives the patient of needed sleep. In all probability it depends upon the action of the biliary acids circulating in the blood upon the cutaneous vasomotor and sensory nerves, as some patients maintain with positiveness that the sense of itching has been preceded by a sensation of coldness in the skin.

Less common cutaneous alterations in association with jaundice are *erythema, urticaria, xanthelasma.*

Not rarely jaundice is attended with **slowing of the action of the heart** and **of the pulse—bradycardia** and **slow pulse.** These likewise are attributable to irritation of the ganglion-cells of the myocardium by the biliary acids circulating in the blood. The pulse has been observed to be as low as 21 beats in the minute.

Among less common phenomena are *disorders of vision,* as, for instance, *yellow vision—xanthopsia*—the development of which is attributed by some to nervous influences, while others explain its occurrence by infiltration of the optic structures of the eye with biliary coloring-matter. At times *hemeralopia* and *nyctalopia* are observed. Besides the blood and the urine, the biliary coloring-matter may appear also in the *sweat* and the *milk,* although it is not found in the saliva, the tears, and the seminal fluid.

Jaundiced patients complain frequently of **anorexia, a bitter taste, insomnia,** and **mental irritability.** The **tongue** is usually coated. The *duration of jaundice* varies with the causes present in the individual case. Chronic jaundice is usually attended with progressive *emaciation,* because the nutrition is impaired and interfered with. Under the same conditions *dissolution of the blood* may occur, so that more or less extensive and numerous hemorrhages may take place into the skin and the various mucous membranes, and also into the retina. Occasionally profound jaundice is attended with nervous manifestations, which have been designated *cholemia.* The patients become stupid and delirious, throw themselves restlessly about in bed, grind their teeth, and are seized with clonic muscular spasm in one or more extremities or throughout the entire body. Death occurs quite frequently under such conditions. The cause of cholemia was formerly thought to be an intoxication of the central nervous system with biliary constituents—according to some with biliary acids, according to others with cholesterin; but at present the tendency is to adopt the view that cholemia depends upon *auto-intoxication*, and that the injurious substances consist in putrefactive alkaloids—ptomains—which are generated in the intestine in consequence of the deranged digestion.

Diagnosis.—The recognition of jaundice is easy, although the clinician should never be satisfied with a diagnosis of jaundice, but in every instance he should determine the causes of this symptom. Under these circumstances considerable difficulty may arise, which can be overcome only by a careful consideration of the etiology and the alterations in individual organs. Icteric discoloration of the skin can be readily recognized only by daylight, and not at all by the light of a lamp. It can be easily differentiated from the **yellowish color of the skin in brunets,** because in the latter the scleræ remain white and the urine presents no change. The skin and the urine may present a yellow color after the use of **picric acid** or **santonin,** and the urine, as in jaundice, may yield a yellow foam, but the color of the urine is a light yellow, and the reaction for biliary coloring-matter is wanting. On the other hand, the urine acquires a reddish color on the addition of potassium hydroxid.

Prognosis.—The prognosis in cases of jaundice depends upon the curability of the causative conditions.

Anatomic Alterations.—In the bodies of those dead of jaundice the icteric color, not only of the skin and the mucous membranes, but also of almost all of the tissues and organs, is striking, the nervous tissues alone remaining unaffected. **Serous effusions** into the pleural cavity, the pericardium, the peritoneal cavity, and in the meningeal spaces present a yellowish, icteric hue. **Chicken-fat clots** in the cavities of the heart likewise present an icteric appearance. The endocardium and the intima of the blood-vessels are stained yellow. The kidneys are characterized especially by their icteric color. The epithelial cells in the convoluted uriniferous tubules are in a state of more or less marked fatty degeneration, and are often stained yellow by biliary coloring-matter. When jaundice has existed for a long time the biliary coloring-matter is precipitated in the form of granules in the epithelial cells. Cylindric collections of biliary coloring-matter also occur in the uriniferous tubules. The **liver** is generally enlarged, and often presents a deep yellowish-black or greenish-yellow appearance. If the jaundice is dependent upon obstruction to the flow of bile by occlusion of the biliary passages, the bile-ducts appear dilated, and in places represent cystic enlargements distended with bile. Under such circumstances there may be connective-tissue hyperplasia and cellular proliferation around the biliary passages. On microscopic examination the liver-cells are found to be saturated with biliary coloring-matter. In cases of long standing the biliary coloring-matter is precipitated in the cells in the form of granules, and less commonly in that of needles or plates. **Enlargement of the spleen** is common. It is important to observe to what point the biliary passages are stained yellow, for the obstruction to the flow of bile must have been seated at the junction between the portion containing and the portion free from bile. This point is often not capable of demonstration in the dead body. At times microscopic examination of the liver furnishes for the first the explanation for the development of the jaundice. Thus, for instance, it has been demonstrated of the jaundice following phosphorus-poisoning that it is dependent upon a catarrhal condition of the smaller intrahepatic biliary ducts.

Treatment.—Although the treatment of jaundice should always be directed to the removal of the causative factors, there are, however, certain rules to be observed in the presence of any variety of the condition. In connection with the **food** it should be a fixed rule to interdict the use of fat (butter, fat meat, fatty sauces, unskimmed milk, yolk of egg). Weak coffee with milk, tea with milk, strained meat-broth, lean meat, good white wine, alkaline and carbonated waters, may be permitted. Daily **evacuation of the bowels** should be secured. Rectal injections of water at ordinary temperature are to be preferred. A single large dose of calomel (0.5—7½ grains) or of a preparation of rhubarb or senna may prove useful. Patients of means with chronic jaundice may be sent to bathing-resorts, among which especially Carlsbad, Marienbad, Homburg, Kissingen, and Tarasp may be recommended. Of medicaments, hydrochloric acid and nitric acid are much employed, and these in any event exert a favorable influence upon the stomach, whose function is generally affected:

℞ Solution of dilute hydrochloric acid,	5.0 : 180.0 (75 minims : 6 fluidounces).
Sirup of raspberry,	20.0 (5 fluidrams).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours after food.

℞ Dilute hydrochloric acid,	
Dilute nitric acid,	each, 2.5 (40 minims);
Sirup of raspberry,	20.0 (5 fluidrams);

Distilled water sufficient to make 200 (6½ fluidounces).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours.

For the relief of the often intolerable *itching of the skin* warm baths may be employed (28° R.—35° C.—95° F.), followed by the application of a carbolated ointment:

℞ Carbolic acid,	5.0 (75 grains);
Wool-fat,	
Lard,	each, 25.0 (6 drams).—M.

For inunction.

Application to the skin of lemon-juice and the administration of potassium bromid internally (2.0—30 grains) have also been recommended. In order to *accelerate the desquamation of the icteric skin* after recovery from jaundice warm baths with soda (100.0—3 ounces) may be given. For the relief of *cholemia* stimulants should be employed, as, for instance, camphorated oil (1.0—15 minims—subcutaneously thrice daily), and active diuresis should be induced by the generous administration of tea and milk in order to eliminate the injurious substances rapidly from the body. If the patients are unable to swallow on account of stupor, rectal injections of tepid milk or physiologic salt-solution (0.75 per cent.) should be given or subcutaneous infusions of saline solution.

HYPOSTATIC LIVER (VENOUS HYPEREMIA OF THE LIVER).

Etiology.—Hypostasis of the liver develops when the flow of venous blood of the liver from the hepatic veins into the inferior vena cava is obstructed. Such a condition occurs in association with **chronic diseases of the heart** and of **the respiratory organs**, and the mechanism generally consists in failure in the function of the right ventricle. As a result a certain amount of blood is left in the right ventricle after each systole of the heart. In consequence the right auricle next becomes imperfectly emptied, then also the superior and the inferior vena cava, and thus are provided the conditions that lead to hypostasis of the liver.

In rare instances hypostasis of the liver develops in consequence of *stenosis of the hepatic veins*. This is dependent at times upon congenital septum-like obstruction, at other times upon acquired thickening and constriction of the vessel-wall. Under such circumstances the stasis is confined exclusively to the liver and the portal circulation, while in the presence of stasis of cardiac origin the entire distribution of the inferior vena cava is involved in the stasis.

Anatomic Alterations.—Hypostasis of the liver is attended with **increase in size** and **marked hyperemia**, so that the organ often exhibits a reddish-black color. On section an oakleaf-like or nutmeg-like appearance is disclosed, the darker portions corresponding to the dilated central veins in the individual lobules of the liver distended with blood. The organ is therefore also designated *cyanotic nutmeg-liver*. On microscopic examination also the dilatation of the central veins and the efferent intralobular capillaries, and their distention with blood, are conspicuous. If the condition has existed for some time, there may readily result, in consequence of the pressure exerted by the dilated blood-vessels, **atrophy and disappearance of the liver-cells**, and the *atro-*

phic nutmeg-liver develops. Under such conditions also hyperplasia of the connective tissue not rarely takes place around the dilated vessels. The consistence of the liver at the same time increases, and in consequence of cicatricial contraction of the connective tissue the liver may even undergo diminution in size, with the formation of nodules upon its surface—the so-called *cirrhotic nutmeg-liver.*

Symptoms and Diagnosis.—If, as in the majority of cases, hypostasis of the liver is dependent upon chronic disease of the heart or of the respiratory organs, the first manifestation of stasis is general **cutaneous edema** in the lower extremities, as the consequences of general stasis are observed earliest in the dependent portions of the body. Symptoms of **hypostasis of the kidneys** (diminution in the amount of urine, which has a high color and a high specific gravity, with a small amount of albumin) also often appear earlier than those of hypostasis of the liver. Patients with *hypostasis of the liver* complain principally of a sense of weight, of pressure, of constriction, and even of slight pains in the region of the liver. On palpation and percussion the liver is found enlarged, and particularly with its lower border projecting in greater or lesser degree beyond the right costal margin. At the same time the consistence of the organ appears increased, although its surface feels smooth. Manipulation of the liver is frequently attended with tenderness. Not rarely **hypostatic catarrh of the biliary passages** develops, with obstruction and **hypostatic jaundice.** The jaundice is commonly not pronounced, and is often confined to the conjunctivæ. If at the same time there is cyanosis of the skin in consequence of the stasis, the jaundiced integument at times acquires a slightly greenish hue—*icterus viridis.* Examination of the liver is rendered difficult, and sometimes even impossible, when marked ascites is present. An attempt should then be made to determine the limits and the consistency of the organ by shock-like palpation in the region of the liver. It is also advisable under such circumstances to examine the liver with the patient in the left lateral or in the supine decubitus. If the stasis can be relieved and the right ventricle can be strengthened, it is often remarkable in how short a time the enlargement of the liver subsides. If, however, the stasis has existed for a considerable time or has recurred frequently, complete involution will no longer take place, obviously on account of the hyperplasia of the connective tissue of the liver. The liver, therefore, remains hard and large. If ascites be present, it will also prove obstinate under such conditions. In the differentiation of hypostasis of the liver from other varieties of enlargement of the liver and from induration of the liver the demonstration of causes of hypostasis is important.

Prognosis.—The prognosis depends upon the possibility of removing the causes of stasis. As a rule, this is possible at first; but subsequently the heart becomes insusceptible to the influence of medicaments, and death results in consequence of excessive general stasis.

Treatment.—The principal remedies consist in **heart-tonics**, particularly digitalis. In general, the treatment is that of weakness of the myocardium (pp. 23 and 24). In the presence of marked tension and pain in the region of the liver from 5 to 10 *wet cups* should be applied.

INFLAMMATION OF THE SEROUS COAT OF THE LIVER (PERIHEPATITIS).

Anatomic Alterations and Etiology.—A distinction is made between *acute* and *chronic perihepatitis*. Acute perihepatitis is attended with the presence of fibrinous deposits upon the serous covering of the liver. Accumulations of pus also may readily take place between the surface of the liver and the diaphragm, and often the pus is encapsulated in several cavities. At times the serosa of the liver is detached from the surface of the organ by small accumulations of pus. Acute perihepatitis occurs most commonly in association with diffuse **peritonitis.** At times it attends **diseases of the liver** extending to the serous covering, as, for instance, abscess, carcinoma, gumma, echinococcus. Injuries in the region of the liver are of less common occurrence. Whether *refrigeratory* (*rheumatic*) *perihepatitis* occurs is at least doubtful. On the other hand, acute perihepatitis may result by extension from adjacent disease, and it may be superadded to **pleurisy** or **pericarditis.** *Chronic perihepatitis* develops at times from an acute perihepatitis, while in other instances it occurs as an independent disorder. It is characterized by thickening of the serous covering of the liver and by band-like or extensive adhesions between the surface of the liver and neighboring structures, particularly the diaphragm. At times the thickening may attain the extent of several millimeters, and appear white and opaque and cut almost like cartilage. If the liver is entirely surrounded by a serosa thus altered, an appearance is created as if the organ were enclosed within a layer of icing, whence the name *mold* or *cast of the liver.* In consequence of contraction of the thickened connective tissue, nodules not rarely form upon the surface, and distortion of the organ results. Frequently the liver assumes a globular shape. Further, the connective-tissue hyperplasia extends here and there into the superficial interlobular spaces of the liver. **Syphilis** is not an uncommon cause for chronic perihepatitis. Hereditary syphilis also at times causes extensive perihepatitic changes in children. At times the disorder develops in the course of *chronic diseases of the liver*, as, for instance, chronic interstitial hepatitis. *Traumatic chronic perihepatitis* occurs in association with constricted liver in the course of the constriction and in the adjacent tissue. *Chronic peritonitis* also may give rise to chronic perihepatitis.

Symptoms and Diagnosis.—*Acute perihepatitis* can be diagnosed with certainty only when a **perihepatitic friction-sound** is generated with the respiratory movements of the liver. This may be appreciable only with the stethoscope, or it may be felt with the hand as the creaking of new leather. It is encountered most commonly over the lower portion of the right side of the chest, although it may be observed also upon the lateral and posterior aspects, or it may be present in these situations and absent anteriorly. At times it can be induced artificially by sliding the abdominal walls upon the surface of the liver. Its duration is susceptible of great variation. At times it is observed for only a few minutes, in other instances for several weeks, now disappearing, but soon reappearing suddenly. Perihepatitic friction is distinguished from pleuritic friction-sounds by the fact that it extends especially toward and may even pass beyond the lower border of the chest. The patients complain principally of **pain in the region of the liver**, which is increased on deep inspiration and on pressure. On percussion it may often be demonstrated that the **respiratory movements of the liver** are restricted, because the patients involuntarily avoid active respiratory movement on account of the pain. Consequently a slight degree of **hypostatic jaundice** develops at times, as active respiratory movement of the liver favors the discharge of bile. *Chronic perihepatitis* also is frequently attended with palpable and audible **friction-phenomena.** Intimate adhesions to surrounding structures prevent the *respiratory displacement of the liver*. If the hepatic veins or the inferior vena cava be surrounded and constricted in consequence of connective-tissue thickening, **ascites** or **general stasis in the veins of the extremities and of the abdomen** develops. At times **stenosis of the portal vein** or of the large **biliary ducts** in the hilus of the liver results, and is followed by incurable ascites or persistent icterus.

Prognosis.—The prognosis of acute perihepatitis is favorable if the causative conditions are not dangerous. The prognosis of chronic perihepatitis also is usually not unfavorable. Stenosis of the inferior vena cava and the hepatic veins, of the portal vein, and the large biliary ducts naturally gives rise to conditions of incurable venous stasis or to chronic jaundice, which finally terminate fatally with progressive asthenia and interference with the movements of the diaphragm.

Treatment.—If pain be severe, hot **cataplasms** over the liver and **cups**, and even **subcutaneous injections of morphin** may be advised. Excessive ascites will require repeated **puncture of the**

abdomen. Chronic jaundice will demand regulation of the **diet** and regularity of **bowel-movement.**

SUPPURATIVE HEPATITIS.

Etiology.—Suppurative inflammation of the liver occurs only when **bacteria** capable of inducing inflammation gain access to the organ. The demonstration of bacteria will be possible only in recent accumulations of pus, as they undergo destruction in the older, so that the abscess becomes sterile. Streptococcus pyogenes, Staphylococcus pyogenes albus and aureus, and Bacterium coli commune have been found in abscesses of the liver.

In abscesses of the liver secondary to dysentery the *amebæ of dysentery* have been observed repeatedly, although it is doubtful whether these are not rather accidental contaminations, the suppuration being really induced by pyogenic cocci. The same statement is applicable to an observation of *flagellates* in hepatic pus.

Any of the vessels of the liver may be the portal of entry for bacteria into the organ; thus the hepatic artery, the portal vein, the hepatic veins, the biliary passages, and in the newborn the umbilical vein. The biliary passages and the portal vein are the most common channels. The *portal vein* is a frequent portal of entry for bacteria into the liver in connection with all **pyemic abscesses of the liver.** These develop when inflammatory processes occur in any organ connected with the distribution of the portal vein, and from which exciting agents of inflammation can enter the portal vein and its intrahepatic ramifications. Among primary inflammatory processes may be mentioned ulceration of the intestinal mucous membrane, in connection with which endemic tropical dysentery particularly has an unfavorable reputation, operations upon and inflammation of the rectum, the uterus, the ovaries, and the tubes, the testicles, ulceration of the vermiform appendix and the stomach, the spleen, the pancreas, etc. Suppurative pylephlebitis may also be responsible for secondary suppuration in the liver. Suppuration in the liver by way of the *biliary passages* occurs especially in the sequence of **gall-stones,** in connection with which the Bacterium coli commune appears to be the exciting agent of the purulent process with especial frequency. Less commonly **parasites in the biliary passages** may be the starting-point for suppuration in the liver.

The *hepatic artery* is the medium for infection of the liver particularly in the presence of **ulcerative endocarditis,** and at times also in association with **pulmonary gangrene** and **putrid bronchitis,** as the thrombi, containing bacteria, invade the distribution of the aorta, and then that of the hepatic artery, finally to become obstructed in the arterial capillaries of the liver, and from this point to excite inflammation of the liver. Bacteria can enter the liver through the *hepatic veins* only by passing in a direction contrary to that of the blood-stream. Experimental investigation has shown that corpuscular elements may pass downward in the inferior vena cava, in opposition to the blood-stream toward the heart, and enter the hepatic veins, and this phenomenon has been accepted in explanation of cases of suppuration of the liver in which hepatic abscesses have developed in the sequence of inflammation and suppuration of the cranium and at the periphery of the body. At the present day such an assumption is, in our opinion, no longer necessary, as bacteria can, without difficulty, if they have gained entrance through the superior vena cava to the right auricle, the right ventricle, and the pulmonary artery, migrate through the pulmonary capillaries, and penetrate the pulmonary veins, the left side of the heart, the aorta, and the hepatic artery. Only if the question arose as to the dissemination of coarse, solid particles would scarcely any other explanation be conceivable than the entrance from the superior into the inferior vena cava and thence into the hepatic veins and their ramifications. In the newborn, infection of the liver sometimes occurs through the umbilical wound or through the intermediation of the *umbilical vein.* Infection of the umbilical wound, in case the mother be suffering from childbed-fever (puerperal septicemia), or infection of the umbilical wound from want of cleanliness in treatment, is the causative factor. It thus appears that suppurative inflammation of the liver may occur at any period of life. At times abscess of the liver results from **injuries** to the organ, even of a blunt character. In other instances it develops in the course of **infectious diseases**, as, for instance, malaria. It is especially widespread in the Orient, in consequence of the frequent occurrence of endemic dysentery. There are, however, cases in which no cause can be demonstrated—so-called *spontaneous abscess of the liver.* An especial group of cases of abscess of the liver is constituted by those varieties that develop in consequence of the *suppuration of tubercles, carcinomata,* or *echinococci of the liver.* Abscess of the liver occurs most commonly in *men.*

Anatomic Alterations.—A distinction must be made between *single* and *multiple abscess of the liver.* Multiple abscesses occur in the liver particularly in relation with pyemic influences. The *size of a liver-abscess* is susceptible of great variation. At times the collection of pus is scarcely visible, while at other times it may be so enormous as to occupy almost an entire lobe. In accordance with the situation of the accumulation of pus a distinction can be made between *superficial* (*peripheral*) and *deep* (*central*) abscesses. Accumulations of pus of some standing are usually surrounded by a connective-tissue capsule, whereas more recent accumulations are in direct contact with the tissue of the liver—*encapsulated* and *non-encapsulated abscesses of the liver.* The pus in many instances resembles ordinary greenish and creamy pus, although at times it acquires a viscid consistency or, from being stained by bile, an appearance of wine-lees. At times, also, it possesses putrid qualities. The liver is as a rule enlarged, and almost always there is likewise *enlargement of the spleen.* If the abscesses of the liver are of pyemic origin, similar collections of pus may be present in numerous other organs.

On *microscopic examination* necrosis of the liver-cells and the adjacent tissue will be found in the structures surrounding the pyogenic bacteria. Next there collect about these small necrotic areas round cells that have probably wandered principally out of the blood-vessels, but have resulted in part also by multiplication of the liver-cells and other cells. A submiliary or miliary abscess is thus formed. By the confluence of adjacent small abscesses larger collections of pus are formed. Even large abscesses not rarely coalesce with one another, as indicated by their multilocular form.

Symptoms and Diagnosis.—In order to be recognized with certainty during life, abscesses of the liver must have attained a certain size and be situated at the surface of the organ. Deeply seated collections of pus are either entirely inaccessible to recognition or can at best be suspected with some degree of probability. The demonstration of a **fluctuating prominence upon the surface of the liver** is decisive in the diagnosis. Although unilocular echinococcus and soft carcinomata or sarcomata of the liver are also attended with fluctuating prominences, **septic constitutional manifestations** are wanting under these conditions. Naturally an abscess of the liver will be accessible to the palpating fingers only when it is in contact with the abdominal wall, and but rarely will it be possible to feel it in the intercostal spaces. If it has developed at the summit of the convexity of the liver, it can be recognized only from the presence of **dulness** on percussion, which abruptly interrupts the course of the upper border of the liver and forms a convex prominence toward the pleural cavity. Under such conditions there may be danger of confounding an abscess of the liver with an **encapsulated pleural empyema** or with a **subphrenic abscess** or with an **echinococcus-cyst.** It will then be necessary to take into consideration the mode of development of the disorder (cough, pleural pain, previous gastric or intestinal disturbance), and to make exploratory puncture of the doubtful accumulation of pus. A fecal odor of the pus is suggestive of a subphrenic abscess, while in the presence of an echinococcus-cyst a clear fluid free from albumin would be expected. At times an abscess of the liver is so closely applied to the abdominal wall that it may be mistaken for an **abscess of the abdominal wall.** The important point in differential diagnosis would then be whether the accumulation of pus moves with the respiratory movement, and this is indicative of abscess of the liver.

The *development of an abscess of the liver* is usually attended with t indefinite symptoms. The **patients complain frequently** of n **in the liver,** which at times extends to the right shoulder and right arm; also, the liver is frequently tender on pressure, in rt throughout an extensive area and in part to an especially arked degree in a circumscribed area corresponding to the situa-on of the abscess. **Enlargement of the liver** then usually takes lace progressively. Frequently **jaundice** develops, but this is by o means a constant symptom. **Enlargement of the spleen** is almost lways demonstrable (infection-spleen). In addition there are signs of general **sepsis**: irregular febrile movement, generally remitting or hectic fever, chills preceding the elevation of temperature, sweats, loss of appetite, progressive anemia, and exhaustion. Should permanent relief not be afforded, death may result from asthenia. At times, however, alarming *complications* may arise in consequence of *rupture of a liver-abscess.* Rupture into the peritoneal cavity is particularly dangerous, as this is generally soon followed by fatal perforative peritonitis. The abscess may, however, rupture also into any of the adjacent organs (pleural cavity, pericardium, lungs, stomach, intestines, urinary passages), and even through the external integument, and at times a natural cure of the abscess has then been observed to take place. A serious occurrence is rupture of the pus into the hepatic veins, the portal vein, or the inferior vena cava, as fatal hemorrhage or general septicemia may result.

The *duration of an abscess of the liver* may extend over a few weeks, several months, and a number of years (up to fifteen), so that a distinction is made between acute, subacute, and chronic abscess of the liver. In addition to rupture, natural cure of an abscess of the liver may take place in consequence of inspissation and calcification, and in the case of small collections of pus, of cicatrization; but these occurrences are extremely rare. Small and deeply situated abscesses of the liver cannot be recognized during life. In some instances scarcely more than the clinical picture of *septicopyemia* is developed, without alterations in the liver itself. At times chills occur at such regular intervals that intermittent fever may be suspected, but malarial plasmodia cannot be found in the blood. Also, if jaundice should develop the diagnosis cannot be established beyond the point of probability, as jaundice may occur in conjunction with pyemic conditions, even apart from suppuration of the liver.

Prognosis and Treatment.—The prognosis of abscess of the liver has been materially improved since time is no longer wasted with internal remedies, but as soon as the condition is discovered it is subjected to surgical treatment. Naturally, the prognosis depends besides upon the nature of the causative factors. The outlook for pyemic abscesses is grave, because they are often multiple, and are, therefore, less readily susceptible to surgical tion and, besides, because the pyemic constitutional state is in itself a most serious condition. An abscess of the liver should be **opened** as soon as the diagnosis is made with certainty, in order to avoid the dangers of rupture. Internal remedies are incapable of causing absorption of pus in the liver. A nutritious diet and alcohol in generous quantities may be advised for the septicopyemic general condition.

CHRONIC INTERSTITIAL HEPATITIS (CIRRHOSIS OF THE LIVER).

Etiology.—Chronic interstitial inflammation of the liver is also known as *contracted liver* and *cirrhosis of the liver*, but both of these designations are inadequate, because chronic interstitial inflammation of the liver does not always give rise to diminution in the size or to contraction, and because, also, the diseased organ does not always present a light-yellow color, for *κίῤῥός* means yellow. The causes for chronic interstitial inflammation of the liver consist in **chemic poisons, bacterial poisons (toxins)**, or **anomalous metabolic products.** It has been known for a long time that excessive indulgence in alcohol is the most common cause for the disorder. Spirit-drinkers are particularly exposed to danger, and the condition has been spoken of as *spirit-drinkers'* or *drunkards' liver*, and the less dilute and the more abundantly spirit is drunk the more readily does the disease develop. Those who partake liberally of food in conjunction with indulgence in spirit may the longer escape the evil effects of their habits. Cirrhosis of the liver occurs much less commonly in those who indulge in wine and beer.

Whether other *irritating articles of food* also are capable of inducing interstitial inflammation of the liver is not definitely known. It has been stated that such a result may be brought about by excessive use of curry. In animals hyperplasia of the interstitial connective tissue of the liver has been induced by long-continued administration of phosphorus. I have observed a similar condition in a girl after *phosphorus-poisoning*.

Cirrhosis of the liver develops occasionally in the sequence of **infectious disease**, as, for instance, after hereditary or acquired syphilis, pulmonary tuberculosis, miliary tuberculosis, and malaria. At times the disease is associated with **alterations in the biliary passages** (calculi, stenosis or occlusion), and under such circumstances infectious factors must be taken into consideration. Possibly the toxins of the colon-bacterium play an important part, as this organism, under the conditions named, is readily capable of gaining access to the biliary passages. Among the *metabolic disorders* that may give rise to chronic interstitial inflammation of the liver *diabetes* and *gout* may be named. Some varieties of chronic interstitial hepatitis are dependent upon *arteriosclerotic alterations in the hepatic artery* and its intrahepatic ramifications, and in the same way as in the kidneys also ***senile contraction of the liver*** may be recognized. Among all of the causative factors excessive indulgence in alcohol is the most important, and this explains the fact that the disease is most common in *men in the lower walks of life*, and that it is more prevalent in some countries than in others. Experience, unfortunately, demonstrates that at times children also are encouraged by their parents to drink spirit, with the result that cirrhosis of the liver develops.

Anatomic Alterations.—Chronic interstitial inflammation of the liver may appear in various anatomic forms, and accordingly **several varieties** of the disease have been distinguished. All, however, agree in the fact that the interlobular connective tissue throughout the entire liver has undergone diffuse and abnormal hyperplasia. Accordingly that variety of circumscribed connective-tissue hyperplasia that takes place about abscesses, new-growths, and echinococci in the liver, and forms a capsule for these, does not belong in this category. Likewise the connective-tissue hyperplasia that develops in connection with conditions of chronic venous stasis and involves at first and principally the dilated intralobular blood-capillaries is not included. Probably every liver in which the connective tissue has undergone morbid hyperplasia appears at first enlarged. Subsequently cicatricial contraction of the connective tissue often takes place, and as a result the *contracted liver* or *atrophic cirrhosis of the liver* develops. If, on the contrary, the liver remains persistently enlarged, the condition is one of *hypertrophic cirrhosis of the liver*. The drunkard's liver is usually one of atrophic cirrhosis, but there are cases in which, although the disease has been of long duration, the liver remains enlarged.

We shall first describe fully *alcoholic cirrhosis of the liver*. In the developed stage of the disease the liver is diminished in size, particularly its left lobe, and the designation contracted liver is therefore appropriate for this condition. The organ is light yellow or light yellowish-brown in color, whence the designation cirrhosis of the liver. The surface of the organ exhibits numerous irregularities and nodules, in consequence of which the disease has acquired the name of *granular atrophy of the liver*. The nodules vary in size between that of a poppy-seed and that of a cherry. The depressions are represented by a grayish, almost translucent, tissue, and they result from contraction of the newly formed connective tissue, while the projecting nodules consist of liver-tissue, which in places presents an almost ochre-yellow color from saturation of the liver-cells with biliary coloring-matter. The capsule of the liver often appears thickened in circumscribed or in extensive areas, and it is at times adherent to adjacent organs, particularly the diaphragm. Also, on section of the organ the nodular appearance of the surface is conspicuous, and likewise here, as upon the surface of the liver, grayish, depressed areas, consisting of newly formed connective tissue, are visible, and between which light-yellow or ochre-yellow colored liver-tissue projects. At times, owing to the dense hyperplastic connective tissue present, the liver has acquired such an indurated consistency that on section with a knife a creaking sound is audible, and accordingly the designation *hardening* or *induration of the liver* seems appropriate. In some cases *thrombosis of the portal vein* develops. The *abdominal cavity* almost always contains transudate. The *spleen* is generally enlarged and hard; the mucous membrane of the stomach and intestines presents the appearances of chronic catarrh.

On *microscopic examination of the liver* the increase in the interlobular connective tissue is conspicuous. Generally several lobules are surrounded by a broad band of connective tissue, and for this reason alcoholic cirrhosis has also been designated *multilobular cirrhosis of the liver*. The connective tissue often presents the appearance of cicatricial tissue deficient in cells, although in more recent areas an abundance of young cells are present, and these are particularly numerous close to the peripheral liver-cells. The *intrahepatic branches of the portal vein* are obstructed or occluded in numerous places in consequence of obliterative endophlebitis. It is assumed that the vascular changes are dependent upon the connective-tissue hyperplasia, and for this reason alcoholic cirrhosis of the liver is designated also *portal cirrhosis*. The *liver-cells* themselves are often in an advanced state of fatty degeneration, and this is probably due in part also to the action of the alcohol, while in other places they have undergone atrophy in consequence of the pressure exerted by the newly formed connective tissue. Often they are stained deeply yellow with biliary coloring-matter. The ascites that almost constantly attends alcoholic cirrhosis of the liver, the hypostatic catarrh of the gastro-intestinal membrane, and the enlargement of the spleen are considered as consequences of *stasis in the portal circulation* dependent upon obstruction of the intrahepatic branches of the portal vein.

An *hypertrophic biliary* or *monolobular cirrhosis of the liver* has recently been distinguished from the alcoholic form of chronic interstitial inflammation of the liver first accurately described by Laennec, and therefore designated Laennec's cirrhosis of the liver. The former is characterized by abnormal enlargement of the liver, with a smooth surface and a bilious and dark-green color. The bands of connective tissue may attain quite considerable proportions, and the liver may then be traversed by grayish bands, which at times may be confounded with neoplastic infiltration. The biliary passages frequently are abnormally dilated and excessively distended with bile. The weight of the liver may become twice or thrice the normal. On microscopic examination the hyperplastic connective tissue will be found generally surrounding individual lobules of the liver (monolobular cirrhosis), and to be the seat of marked increase and ramification of the smaller biliary passages, from which the connective-tissue hyperplasia appears to have arisen (*biliary cirrhosis of the liver*). The newly formed connective tissue exhibits no tendency to undergo cicatricial contraction, and therefore the liver does not become reduced in size.

Syphilitic cirrhosis of the liver has been designated also *monocellular cirrhosis of the liver*, because the connective-tissue hyperplasia occurs about individual liver-cells and causes their destruction by pressure. The liver is often greatly changed in shape, one or more portions of the organ becoming detached from the main body by bridges of connective tissue, and the liver is thus converted into a multilobulated organ—*syphilitic lobulated liver*.

Little is as yet known with regard to *senile* or *arteriosclerotic cirrhosis of the liver*, which resembles the alcoholic variety of the disease and is attended with contraction after previous enlargement. Microscopically the extensive obliterative endarteritis in the intrahepatic branches of the hepatic artery is a conspicuous feature.

Symptoms and Diagnosis.—The several anatomic varieties of cirrhosis of the liver correspond also with various clinical pictures, and we shall describe these with the qualification that *typical conditions* will be considered. The distinctive symptoms of *alcoholic cirrhosis of the liver* are usually preceded by manifestations of chronic gastro-intestinal catarrh, which likewise is dependent upon excessive indulgence in alcohol. The patients become more and more emaciated, lose their appetite, and acquire a peculiar **grayish-yellow color of the skin.** The scleræ generally are distinctly jaundiced. Marked icteric discoloration of the skin and mucous membranes does not occur, as a rule. In some cases **disagreeable sensations of tension, of pressure, and even of pain in the region of the liver** occur, while in others attention is first attracted to the disease by increase in girth in consequence of accumulation of fluid in the abdominal cavity. If opportunity be afforded to examine the **liver** at an early stage of the disease, it will be found to be enlarged, with its lower border at the level of the umbilicus, and even lower. At the same time the hardness of the organ is notable, and this may be distinctly recognized by palpation of the lower margin of the liver. Gradually the liver undergoes progressive diminution in size, and its lower border will be found above the right costal margin. If the abdominal walls are thin and deficient in fat, the trained hand may succeed in detecting nodular irregularities upon the surface of the liver. The examination of the liver is sometimes attended with insurmountable difficulty from the accumulation of fluid in the abdominal cavity, causing distention of the abdominal walls and displacing the liver upward into the concavity of the diaphragm. At times the information desired can be gained through percussion and palpation to a certain degree by practising shock-like palpation in the dorsal decubitus, or percussion and palpation with the patient occupying the left lateral decubitus or the prone position.

Dropsy of the abdominal cavity—ascites—is quite a constant symptom of alcoholic cirrhosis of the liver, and is dependent upon stenosis and partial occlusion of intrahepatic branches of the portal vein, and the resulting increase of blood-pressure in the trunk of this vein. The abdominal walls are not rarely distended to the utmost, and the heart and the lower margins of the lungs, together with the diaphragm, may be greatly displaced upward. The **umbilicus** not rarely forms a marked projection forward, and in transmitted light appears translucent because the umbilical hernia contains serous fluid from the abdominal cavity. Not rarely points of rupture in the cutis are observed upon the lower half of the abdomen in the form of striæ. Often excessively distended and markedly tortuous **cutaneous veins** in the abdominal wall attract attention. Usually the trunk of each inferior epigastric vein ascends from the middle of Poupart's ligament, and sends off various ramifications at the level of the umbilicus. These unite with branches of the superior epigastric veins, which generally disappear at the level of the nipple and in the direction of the axillary cavity. Obviously these conditions are dependent upon the venous collateral circulation that develops because the ascitic fluid rests upon and compresses the inferior vena cava.

An uncommon variety of venous collateral circulation in cases of cirrhosis of the liver is known as the *caput Medusæ—cirsomphalos*. This consists in the presence of greatly dilated veins surrounding the umbilicus, and constituting a sort of cavernoma. The vessels are branches of the epigastric veins, which have entered into anastomosis with the patulous umbilical vein in the round ligament of the liver.

In contradistinction from hypostatic ascites, it is distinctive of the ascites of alcoholic cirrhosis of the liver that the extremities are free from cutaneous edema. Only when the ascites, by reason of its weight, causes considerable stenosis of the vena cava will hypostatic edema develop; but this is characterized by the fact that it is slight as compared with that of marked ascites. At times cachectic edema of the skin, or edema of the extremities on one side, develops in consequence of marantic thrombosis of the femoral vein. The **spleen** is almost always enlarged, although excessive ascites often prevents recognition of this condition. Recently attention has been called to the fact that the old view, according to which the enlargement of the spleen is due to stasis in the portal vein, appears not to be correct, because enlargement of the spleen at times occurs so early that it is more probably correct that the enlargement of the spleen and that of the liver are dependent upon identical causes. If the excessive indulgence in alcohol has already given rise to **chronic gastro-intestinal catarrh,** this becomes materially aggravated in consequence of portal cirrhosis of the liver, because the obstruction and occlusion of the intrahepatic branches of the portal vein give rise to stasis in the veins of the stomach and the intestine. At times **hemorrhoids** develop. If the stasis becomes excessive, there may be extensive hemorrhages in the mucous membrane of the stomach, and **vomiting of blood—hematemesis.** At times, however, blood that is vomited may come from *varicose dilatations of the veins in the lower portion of the esophagus.* Death from hemorrhage may take place within a short time from uncontrollable bleeding. The *duration of alcoholic cirrhosis of the liver* is, as a rule, more than a year (up to five years). But rarely is an acute course of a few weeks observed. The *course* of the disease is usually afebrile. At times, however, febrile movement occurs, the cause of which is unknown—*hepatic fever.* In some cases death results from *asphyxia* and *cardiac paralysis* if the accumulation of fluid in the abdominal cavity has become so considerable that the lungs and the heart are unduly displaced upward. Other patients die amid signs of *progressive asthenia.* Under such conditions symptoms of dissolution of the blood appear upon the skin and the mucous membranes. The occurrence of fatal hemorrhage from the dilated esophageal veins has already been mentioned.

The *diagnosis of alcoholic cirrhosis of the liver* is easy if the three main symptoms—a small, hard, nodular liver, enlargement of the spleen, and ascites—are present, particularly if the patient is admittedly a spirit-drinker and presents a sallow, slightly icteric tint of the skin. Should, however, marked ascites prevent examination of the liver and the spleen, the condition may be confounded with **thrombosis of the portal vein, and tuberculous, carcinomatous, and chronic-serous peritonitis.** Under such conditions the most reliable procedure is to remove the ascites by puncture of the abdominal cavity, because subsequently the liver and the spleen become accessible often with surprising distinctness immediately after the puncture, and are susceptible of examination. At times, naturally, the liver remains in the concavity of the diaphragm in spite of abdominal puncture, generally in consequence of perihepatitic adhesions to the diaphragm. It should then not be forgotten that thrombosis of the portal vein usually develops in the sequence of inflammatory and ulcerative processes in the stomach, intestine, and genito-urinary apparatus; that carcinomatous inflammation of the peritoneum occurs in advanced life, and is associated with cachexia; and that tuberculous peritonitis is generally secondary to tuberculosis of the lungs, intestines, or mesenteric glands, and is usually associated with pain and febrile movement, but without icterus. At times **pericardial adhesions,** with myocardial weakness and stasis, particularly in the veins of the peritoneum, constitute a source of great diagnostic difficulty. The decision will then depend principally upon the demonstration of the previous presence or absence of pericarditis. The typical picture of *hypertrophic* or *biliary cirrhosis of the liver* is produced by that variety of the disease which has been designated also *hypertrophic cirrhosis of the liver with jaundice*. In this disorder the *liver* is greatly enlarged, and its lower border often extends considerably below the level of the umbilicus. Also its upper border may be displaced upward. At the same time the organ is characterized by increased resistance and hardness, although its surface appears smooth, and the liver does not undergo diminution in size. The *spleen* also usually exhibits considerable enlargement. *Ascites* is not present, but on the other hand *jaundice* is marked. The patients complain principally of a sense of pressure and of tension in the region of the liver and of derangement of gastro-intestinal activity. The disease usually pursues a *slow course*, and at times may persist for more than five years. *Progressive emaciation*, at times also cholemic manifestations, usually lead to death.

In two cases I have observed the clinical picture of hypertrophic cirrhosis of the liver with jaundice in which post-mortem examination disclosed the existence of *pseudoleukemia*. Enlargement of the liver and of the spleen had resulted from pseudoleukemic new-growths, and the profound icterus from the pressure exerted by enlarged lymph-glands upon the hepatic ducts in the transverse fissure. Between portal and biliary cirrhosis of the liver there are various relations and gradations, so that a whole series of varieties of cirrhosis of the liver has been described. The following schematic enumeration is presented :

I. *Alcoholic or portal cirrhosis of the liver.*
  - *a.* With atrophy of the liver, ascites, and enlargement of the spleen ;
  - *b.* With hypertrophy of the liver, ascites, and enlargement of the spleen ;
  - *c.* With jaundice.

II. *Hypertrophic or biliary cirrhosis of the liver, with jaundice.*

III. *Biliary cirrhosis of the liver, with consecutive atrophy.*
  - *a.* With enlargement of the spleen ;
  - *b.* Without enlargement of the spleen.

In one of my patients a monolobular form of cirrhosis of the liver was suddenly superadded to a preëxisting multilobular cirrhosis and caused death in the second week of the disease, being attended with enlargement of the spleen, but without jaundice and ascites ; and in another in which death resulted from cholemia, there was found on macroscopic examination the typical appearances of hypertrophic portal cirrhosis of the liver, while microscopic examination disclosed a most beautiful example of monolobular cirrhosis of the liver. Ascites was not present. The classifications suggested therefore scarcely indicate more than that every case of cirrhosis of the liver may possess peculiarities of its own, and should be carefully examined. Upon what these peculiarities depend has, however, not yet been explained.

Syphilitic cirrhosis of the liver is characterized especially by the constricting off of individual portions of the liver. It may be readily confounded with constricted liver and with tumors of the liver. Little is known with regard to *senile* or *arteriosclerotic cirrhosis of the liver.* It develops insidiously in elderly persons who perhaps may always have passed temperate lives, the liver being large and hard, and subsequently undergoing reduction in size. Ascites occurs and death results from excessive asthenia.

Prognosis.—The prognosis of cirrhosis of the liver is unfavorable, for there is no remedy capable of causing disappearance of the hyperplastic connective tissue, and, besides, the disease exhibits an undeniable tendency to progress. As a rule, the patients are at so advanced a stage of the disease when they come under observation for treatment that the outlook for therapeutic success is small. Nevertheless, isolated observations have been made in which it has been possible to check the progress of the disease, and to relieve distressing symptoms permanently, particularly the ascites.

Treatment.—In the treatment **causal therapy** plays an important part. The patients should be weaned from the use of alcohol, or if syphilis be the etiologic factor, *potassium iodid* (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily), *mercurous chlorid* (0.5—7½ grains; 1 such powder thrice weekly), or *mercurial ointment* (5.0—75 grains—to be used by inunction daily for 25 or 30 days) should be prescribed. In cases of arterio-sclerotic cirrhosis of the liver the employment of potassium iodid may be recommended. Among **symptomatic remedies** *potassium iodid* and *mercurous chlorid* occupy first place. A **milk-diet** should be observed (1 or 2 quarts of thoroughly boiled milk in small amounts at intervals of from one-quarter to one-half an hour throughout the day), as milk is an easily digestible and nutritious article of food, and it also stimulates diuresis. Local treatment will be required with especial frequency for the excessive *ascitic accumulation.* Generally little can be accomplished with diuretics, laxatives, and diaphoretics. The most reliable measure is **abdominal puncture.** As a rule, the fluid naturally soon reaccumulates, but there is no objection to repeating the puncture from time to time. It is properly becoming more commonly the practice not to reserve puncture as a last resort, but to employ it early in order to avert threatening asphyxia. At times reaccumulation of the fluid does not take place after puncture, and recovery ensues. Courses of treatment at **springs** should be prescribed only when ascites is absent or inconsiderable, so that the patient can move about. Carlsbad, Marienbad, Homburg, Kissingen, and Tarasp especially may be recommended.

ACUTE YELLOW ATROPHY OF THE LIVER.

Etiology.—Acute yellow atrophy of the liver is an exceedingly [illegible] attended with rapid fatty degeneration of the liver-cells, with their disintegration and absorption, so that within a few days the liver becomes greatly diminished in size. The disease develops generally under the influence of **bacterial poisons** (**toxins**) and perhaps also of **putrefactive alkaloids** (**ptomains**), although little of a definite nature is known in this connection. It has been maintained further that certain chemic poisons (phosphorus, fungous poisons) may cause acute yellow atrophy of the liver, but under such circumstances there is probably resemblance rather than identity in the anatomic and clinical pictures. At times acute yellow atrophy of the liver develops in the sequence of **infectious diseases,** as, for instance, typhoid fever, pharyngeal diphtheria, enteritis, and syphilis. At other times the disease occurs as a complication of other **diseases of the liver,** as, for instance, cirrhosis and fatty liver. Occasionally small endemics of acute yellow atrophy of the liver have been observed in barracks, so that the suggestion of an **independent infectious disease** has been raised. It is known that the disease occurs with relative frequency during **pregnancy,** and with particular frequency between the fifth and the eighth month, and less commonly in association with parturition. Under such circumstances ptomains may be the exciting agents. Often a definite cause cannot be demonstrated—*cryptogenetic atrophy of the liver.* Occasionally fright, emotional disturbances of other kind, and alcoholism are assigned as causes. Experience has shown that acute yellow atrophy of the liver occurs *more commonly in women* than in men. Although the disease is most common at the vigorous period of life (from 25 to 45), on the other hand cases have been observed also in children and the aged.

Anatomic Alterations.—On opening the abdominal cavity the **liver** is often not at all visible from having fallen back toward the vertebral column, and being covered in front by loops of intestine. The organ has often lost more than half in size and weight. The capsule of the liver, therefore, is wrinkled. The consistence of the organ is diminished, so that it can almost be shaken to and fro. On section the liver presents a more or less marked ochre-yellow color. In addition areas stained red are present, resulting from the ochre-yellow areas. Red markings are observed particularly in the left lobe of the liver, in which the disease is prone to begin.

On *microscopic examination* attention is attracted particularly by the marked fatty degeneration of the liver-cells. The cells undergo disintegration, the masses of fat are partially absorbed, and eventually there remains a striated empty structure free from liver-cells. The blood-vessels also undergo fatty degeneration. The interlobular biliary passages stand out with marked distinctness, and appear hyperplastic. Cellular proliferation and round-cell accumulation also take place in the interlobular connective tissue, and cirrhosis of the liver has been observed to develop in the sequence of acute yellow atrophy. At times, but by no means constantly, bacteria have been found in the liver (Streptococcus pyogenes, Staphylococcus pyogenes albus, pneumoniacoccus, Bacterium coli commune). If the liver has been exposed to the air for a while, a frost-like deposit at times takes place upon its surface, and this is found upon microscopic examination to consist of needles of tyrosin and spheres of leucin. In all probability acute yellow atrophy of the liver consists in a degenerative process involving the liver-cells, and to which secondarily inflammatory alterations (proliferation of the biliary passages, connective-tissue hyperplasia) are superadded. The remaining organs are generally characterized by their marked **icteric hue.** In addition, **extravasations of blood** occur in the viscera. Usually the glandular cells of the stomach, the intestine, the pancreas, the convoluted urinary tubules, often also the muscle-fibers of the myocardium and of the voluntary muscles, are in a state of fatty degeneration. The **spleen** and the **portal** and **retroperitoneal lymph-glands** are enlarged and softened.

Symptoms, Diagnosis, and Prognosis.—Acute yellow atrophy of the liver often sets in with **prodromes,** consisting in symptoms of gastro-intestinal derangement. Then **icteric discoloration of the skin and of the mucous membranes** takes place, and this attains the highest possible degree of intensity within a short time. Soon symptoms are superadded of a condition that is designated *cholemia,* and which has recently been attributed to auto-intoxication with abnormal metabolic products. Consciousness becomes more and more obscured, delirium develops, and the patients groan and often throw themselves about restlessly in bed, and from time to time exhibit clonic muscular contractions in individual extremities or in all of the muscles. Often symptoms of **blood-dissolution** further appear, and there may be hemorrhages beneath the skin and the various mucous membranes. Amid progressive loss of consciousness the disease generally terminates fatally in the second week, although at times it may be protracted for from six to fourteen weeks.

In the diagnosis of acute yellow atrophy of the liver the **changes in the liver and in the urine** are particularly important. On percussion, and to a certain degree also on palpation, the **liver** will be found from day to day to be growing smaller in size. The dulness yielded by the left lobe disappears earliest, and eventually the entire area of hepatic dulness may be reduced to a narrow band. The liver-dulness may even be wholly wanting, and be replaced by an area yielding a tympanitic note. The diminution in the area of liver-dulness progresses gradually from below upward. Pressure over the liver is in conspicuous degree painful. Diminution in the size of the liver may be simulated by the presence of intestine—

as, for instance, the transverse colon greatly distended with gas—between the surface of the liver and the abdominal and thoracic walls; but generally such a condition is only a transient one, so that within a short time the liver-dulness is completely restored Besides, the liver-dulness can frequently be elicited by pressing the pleximeter firmly upon the abdominal wall, thus flattening the transverse colon upon the surface of the liver. Also, in the presence of **free perforative peritonitis** disappearance of liver-dulness takes place when the gas that has escaped from the stomach or the intestine into the abdominal cavity insinuates itself between the liver and the thoracic and abdominal walls, but such a condition develops suddenly, and is, or at least may be, unattended with icterus, while peritonitic pain, abdominal distention, and the like predominate.

The **urine** is usually passed in small amounts, possesses a deep-brown, icteric hue, and generally contains albumin, fibrinous tube-casts, epithelial cells from the uriniferous tubules and the bladder and round cells. Of particular importance is the occurrence of **needles of tyrosin** in the urinary sediment, and which usually lie together in sheaf-like arrangement, and of **spheres of leucin**, several of which are often coherent, and which are characterized by their double contour. Usually both of these substances are precipitated spontaneously. Should this not be the case, they can be precipitated by adding acetic acid to a drop of urine upon a slide, and permitting evaporation to take place. It may be remarked incidentally that leucin and tyrosin occur in the urinary sediment also in association with other severe varieties of jaundice—as, for instance, following phosphorus-poisoning—and at times also in the course of severe infectious diseases (small-pox). The amount of urea in the urine will be found diminished. Of abnormal urinary constituents, sarcolactic acid has further been isolated. All of these facts indicate that the metabolism is profoundly altered in cases of acute yellow atrophy of the liver. The disease is generally unattended with fever, and almost invariably terminates fatally with progressive asthenia and loss of consciousness. The prognosis is therefore hopeless.

Treatment.—**Causal treatment** is applicable only when syphilis is an etiologic factor, and under such conditions inunctions of *mercurial ointment* (5.0—75 grains) may be employed, and *potassium iodid* (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—a tablespoonful—thrice daily) administered. The **symptomatic treatment** consists in the employment of *stimulants*, as, for instance:

℞ Camphorated oil, 10.0 (2½ fluidrams).
Dose: 1 c.c. (15 minims) subcutaneously thrice daily.

The **administration** of from 1 to 2 quarts of boiled **milk** daily is **advisable in order** to increase the secretion of urine, and thus hasten the elimination of possible toxic metabolic products from the body. It may also be advantageous to stimulate the secretion of urine by permitting 500 c.c. (1 pint) of lukewarm physiologic **salt-solution** (0.75 per cent.) to flow into the rectum or beneath the skin several times daily.

FATTY OR ADIPOSE LIVER.

Etiology.—Fatty liver is only of subordinate clinical significance. A distinction must be made between **cachectic, toxic, infectious fatty liver,** and that due to **over-eating.** *Fatty liver due to over-eating* occurs in association with obesity, and develops usually in persons who eat a great deal, partake excessively of carbohydrates and of alcoholics, and indulge insufficiently in physical exercise. *Cachectic fatty liver* develops in the sequence of loss of blood or of discharges (protracted suppuration) and debilitating diseases (pulmonary tuberculosis, carcinoma, chlorosis, pernicious anemia, leukemia, etc.). *Toxic fatty liver* occurs in the train of certain intoxications, among which poisoning with phosphorus, arsenic, and mercury may be particularly mentioned. *Infectious fatty liver* is that variety that develops not rarely in the sequence of acute infectious diseases. Fatty liver occurs further in association with **venous stasis** in the form of the fatty nutmeg-liver, and more especially locally in connection with **diseases of the liver** in the neighborhood of carcinomatous abscesses and cirrhosis of the liver. A variety of physiologic fatty liver occurs in *infants.* It is a form of fatty liver due to over-feeding, and is dependent upon the use of milk containing much sugar in conjunction with insufficient bodily activity.

Anatomic Alterations.—In cases of well-developed fatty liver the organ is enlarged and its margins blunt. It is light gray or yellowish gray in color. On section of the liver a fatty deposit adheres to the blade of the knife. On microscopic examination the liver-cells are found filled with large and small fat-globules. In addition some of them not rarely contain considerable biliary coloring-matter. A distinction has been made between **fatty infiltration** and **fatty degeneration of the liver,** accordingly as the fat in the liver-cells has been deposited through the intermediation of the blood or has developed in the liver-cells themselves from disintegration of their protoplasm. Probably the fatty liver due to over-feeding is a variety of fatty infiltration; while the cachectic, toxic, and infectious forms, are, on the contrary, varieties of fatty degeneration. The view that fatty infiltration and fatty degeneration are distinguishable from each other by the fact that in cases of the former the liver-cells are filled with large fat-globules, while in cases of the latter, on the contrary, the liver-cells are filled with numerous small and minute fat-globules, is not correct. It has been pointed out that the differentiation between the two conditions can be made by chemic means, inasmuch as in the presence of fatty infiltration the amount of water in the liver is reduced (from 70 to 50 per cent.), because the fat has displaced the water of the liver-cells; while in the presence of fatty degeneration the amount of water remains unchanged. Should fatty liver be an associated manifestation of general obesity, unusually large amounts of fat are found also in other organs and structures (subcutaneous connective tissue, epicardium, mesentery, epiploic appendices).

Symptoms, Diagnosis, and Prognosis.—Patients with *fatty liver due to over-eating* not rarely complain of a sense of tension and of constriction in the region of the liver, and on palpation and percussion this organ will be found enlarged, resistant, smooth, with blunt borders, and at times also tender on pressure. The remaining symptoms of general obesity will generally indicate that the manifestations just mentioned are dependent upon fatty infiltration of the liver. To what extent digestion and metabolism are thereby deranged is not definitely known. The prognosis depends upon whether the general obesity can be corrected or not. *Cachectic, toxic,* and *infectious fatty liver* may be wholly unattended with symptoms.

Treatment.—The treatment of fatty liver consists in the treatment of the fundamental disorder, as, for instance, of that variety due to over-eating, in strict regulation of the diet.

AMYLOID LIVER.

Etiology.—Amyloid degeneration of the liver may develop as a result of **wasting discharges** and **cachectic states** of all kinds, as, for instance, protracted suppuration, particularly of bones and joints, of the kidneys and the pelvis, pleural empyema, chronic diarrhea, pulmonary tuberculosis, malarial and syphilitic cachexia. In rare instances it is impossible to ascertain the cause, so that the disease gives the impression of being an independent disorder. The condition occurs at all periods of life, and in children particularly in the sequence of scrofulous—thus actually tuberculous—suppuration of the lymphatic glands, bones, or joints.

Anatomic Alterations.—In cases of well-developed amyloid degeneration the liver is large, smooth, and hard—almost as if frozen. Its weight may be increased threefold. On section the margins exhibit an almost translucent aspect, and upon the cut surface the liver-substance can be scraped with the knife as if the organ were frozen. If tincture of iodin, or, still better, Lugol's solution (pure iodin, 0.5—7½ grains; potassium iodid, 5.0—75 grains; distilled water, 200.0—6½ fluidounces), be poured upon the cut surface, the liver becomes stained **dark brown or** mahogany-brown in the amyloid areas.

On *microscopic examination* of the liver the portions involved in amyloid degeneration can be recognized from their clear, waxy luster. They appear swollen and structureless, but, above all, they yield the **amyloid reaction,** being stained brown with Lugol's solution. If the sections are then placed in a watch-glass containing dilute sulphuric acid, they assume a bluish or bluish-violet tint. Methyl-violet gives the areas involved in amyloid degeneration a bright-red tint, and they are thus sharply differentiated from the adjacent healthy tissue stained blue.

Careful *histologic examination* discloses that the walls of the blood-vessels are first involved in the process of amyloid degeneration, and primarily the walls of the hepatic arteries, subsequently those of the interlobular capillaries and the branches of the portal vein. The liver-cells generally remain exempt, and according to some observers permanently so. *Chemic examination of the amyloid material* discloses that it contains nitrogen, and is a proteid substance. The source of this abnormal metabolic product is unknown. In all probability the amyloid substance is generated locally, for not rarely in the application of the amyloid test appearances are encountered in which a gradual transition in color is observed to take place from healthy to amyloid tissue. Some observers, however, assume that the amyloid substance is brought to the liver from without.

If the amyloid degeneration of the liver is not well developed, it can be recognized only with the aid of the reactions mentioned. Almost always other organs will also be found involved in the degenerative process, particularly the spleen, the adrenal bodies, the kidneys, and the mucous membrane of the intestine.

Symptoms, Diagnosis, and Prognosis.—Amyloid degeneration of the liver will be susceptible of diagnosis only when the liver is large, hard, and smooth; if the condition has been preceded by diseases that experience has shown are frequently followed by amyloid degeneration, and if enlargement and induration of the spleen, edema of the skin, and marked albuminuria and diarrhea indicate simultaneous amyloid degeneration of the spleen, the kidneys, and the intestine. Amyloid liver is not attended with jaundice unless the biliary passages be obstructed by enlarged lymphatic glands in the transverse fissure. The patients are usually pallid in consequence of the conditions that are responsible for the amyloid disease. Recovery can scarcely be expected. Death occurs most commonly from progressive exhaustion.

Treatment.—In the presence of amyloid disease preparations of *iron,* and particularly **iron iodid,** are generally prescribed, although these probably exert a useful influence upon the accompanying anemia rather than upon the amyloid condition. In a

prophylactic way it is important to suppress chronic suppuration, diarrhea, and wasting discharges of all kinds as rapidly as possible.

CARCINOMA OF THE LIVER.

Etiology.—Carcinoma of the liver is an extremely *common disorder*. Almost invariably the condition is one of **secondary carcinoma.** Cases of primary carcinoma of the liver are exceedingly rare. Most commonly secondary carcinoma of the liver develops in connection with *carcinoma of the stomach*, the new-growth extending directly from the stomach to the liver. Frequently, however, portions of the new-growth are conveyed from the stomach to the liver as emboli through the blood-vessels, and thus give rise to secondary carcinoma in the liver. In addition to the stomach, the *uterus*, the *rectum*, the *esophagus*, and the *mammary gland* particularly are often the seat of primary carcinoma, which is often followed by secondary invasion of the liver. It should be pointed out, further, that secondary carcinoma of the liver develops at times in the sequence of *carcinoma of the gall-bladder*. Often biliary calculi are found in the gall-bladder, so that it is reasonable to believe that these may act as the exciting cause of the new-growth in the gall-bladder. Naturally, also, they may have developed subsequently in the diseased gall-bladder. Carcinoma of the liver occurs *more commonly in women* than in men. It usually develops after the fortieth year of life, and rarely earlier.

Anatomic Alterations.—Carcinoma of the liver occurs in two varieties—namely, **circumscribed** and **infiltrating.** *Circumscribed carcinoma* forms well-defined nodules that range in size from being scarcely visible to that of a man's head. The number of nodules varies greatly. At times the liver is so largely occupied by them that but little normal hepatic tissue is left. At the same time the size and the weight of the organ may reach more than six times the normal. Carcinomatous nodules situated at the surface of the liver often exhibit a depression or so-called umbilication. On section carcinomatous nodules usually present a juicy, whitish, or yellowish-white medullary structure, from which carcinomatous juice or milk can be scraped with a knife. At times carcinomatous nodules are conspicuous for an abundance of vessels distended with blood, so that they present a reddish mottled appearance ; blood-lakes at times form in consequence of rupture of blood-vessels. Carcinomatous nodules may acquire a grayish or slaty color from the presence of pigment in large amount, and the condition is then designated pigmentary carcinoma. As in other organs, also in the liver, a distinction is made between fibrous carcinoma (scirrhus), medullary carcinoma, and colloid carcinoma (alveolar carcinoma), accordingly as the new-growth contains much or little fluid or cavities filled with gelatinous or isinglass-like material.

On *microscopic examination* the carcinoma is found to consist of masses of epithelioid cells with large nuclei separated from one another by a connective-tissue framework. These cells originate in part from the epithelial cells of the biliary passages, in part from the liver-cells.

The tissue of an *infiltrating carcinoma* of the liver involves the interlobular connective tissue without sharp limitation, so that unless microscopic examination be made confusion with cirrhosis of the liver is possible. This variety of carcinoma occurs much less commonly than nodular carcinoma.

Symptoms and Diagnosis.—In the diagnosis of carcinoma of the liver the demonstration of **globular projections** from the surface of the liver is almost of decisive significance. Such a condition is naturally possible only when the new-growths are situated upon the surface of the liver. Carcinomata situated in the central portions of the liver are not accessible to diagnosis. Under favorable conditions they may at best be suspected from the fact that the liver on palpation and percussion exhibits deep tenderness in circumscribed areas. As a rule, the carcinomatous projections from the surface of the liver can be detected only by palpation, and but rarely can they also be seen if the abdominal walls are thin and deficient in fat. At times a depression or umbilication can be felt upon the surface of the growths, and in exceptional instances I have even seen this. The neoplastic nodules are often exceedingly tender on palpation and on percussion. At times they are so soft as to exhibit fluctuation. Over highly vascular carcinomata cardiac-systolic vascular murmurs may be audible. Of diagnostic significance is the **respiratory mobility** that the nodules exhibit together with the organ in which they are seated. Only when the liver has become greatly increased in size will the vigor of the diaphragm be insufficient to transmit the respiratory movement to the liver. With a liver the seat of extensive carcinomatous degeneration, not alone may the upper boundary of dulness begin unusually high, but also the lower boundary may extend into the small pelvis. Perihepatitic adhesions of the liver to the abdominal wall and to adjacent abdominal viscera may also prevent respiratory displacement of the liver. At the beginning of the disease it is highly important to palpate most carefully the lower border of the liver throughout its entire extent, as it is just in this situation that carcinomatous nodules may be readily and distinctly felt. Naturally, in doubtful cases the surface of the liver will be examined repeatedly for projections. It is often possible in the course of the disease to detect newly forming nodules and to follow the growth of those already formed.

In the *differential diagnosis* it is important in the first place to determine that a tumor in the region of the liver is situated in that organ, and in the next place to decide as to its carcinomatous nature. In connection with the first point the respiratory displacement of the tumor is important, as this occurs in addition only in connection with tumors of the spleen, and generally not with tumors of the stomach, the kidneys, the intestine, or the mesentery. Besides, it should be noted whether the tumor is situated in the area of hepatic percussion-dulness, and whether it forms a continuous whole with this. The carcinomatous nature of a tumor of the liver will be rendered highly probable if the new-growth has developed spontaneously after the fortieth year of life, and has been attended with rapid emaciation. As compared with **abscess of the liver,** it should be noted that fluctuation is the rule when the accumulation of pus is situated superficially, while it is the exception with carcinoma of the liver. Abscess of the liver is usually attended with febrile movement, while carcinoma of the liver is generally unattended with elevation of temperature. In addition, the etiology must be taken into consideration: carcinoma develops spontaneously, while abscess is preceded by traumatism, gall-stones, ulceration of the bowel or of the genitalia, or septicopyemia.

Unilocular echinococcus of the liver likewise gives rise to a projection from the surface of the liver, but, in contradistinction from that due to carcinoma, the former is attended with fluctuation, yields hydatid fremitus, and on exploratory puncture gives exit to clear watery fluid free from albumin, in which microscopic examination discloses at times the presence of echinococcus-hooklets and echinococcus-scolices.

Great difficulty may be encountered in the differentiation between carcinoma of the liver and **multilocular echinococcus of the liver,** as both diseases are attended with nodular enlargement of the liver, jaundice, and enlargement of the spleen. Multilocular echinococcus of the liver is an exceedingly rare disease, and, therefore, in doubtful cases carcinoma of the liver will always be the more probable condition. The probability will be all the greater should the patient be of an age at which carcinoma is common and have become cachectic within a short time. In the differential diagnosis of carcinoma of the liver it is always of especial importance if primary carcinoma be found in any other viscus.

Gummatous nodules in the liver are not tender on palpation, and there must have been a history of previous syphilis. These tumors diminish and disappear upon treatment with mercurials and iodids.

Should the liver be enlarged but present a smooth surface, the diagnosis of carcinoma of the liver will always be involved in doubt. Naturally, suspicion should invariably be aroused if jaundice develops in an elderly person without demonstrable cause, and if in the course of about four weeks this does not diminish, but rather increases in intensity, and if at the same time there should be rapid loss of strength. Jaundice is an exceedingly common, if not a constant, symptom of carcinoma of the liver, but I have noted its absence in a number of instances in which it would be most readily expected, namely, in the presence of carcinoma of the gall-bladder with extension to the liver.

The *development of jaundice* in cases of carcinoma of the liver is explained by biliary stasis due to stenosis of the biliary passages in consequence of pressure exerted by the carcinomatous nodules. At times jaundice is dependent upon carcinomatous degeneration and enlargement of the portal lymphatic glands, with pressure upon the hepatic duct.

The **stools** are frequently deficient in bile and clay-colored, though at times they retain their natural color, because the bile that enters the intestine is still sufficient to discolor the fecal matter. It may also happen that periods in which the stools are free from bile alternate with those in which bile is present. A restoration of the lumen of the biliary passages is possible from the reduction in size of the neoplastic nodules in consequence of fatty degeneration, so that the pressure exerted by them on adjacent structures varies. Among other sequelæ of jaundice the patients often suffer in marked degree from **itching of the skin—cutaneous pruritus**—which especially disturbs rest at night and contributes materially to exhaustion. The skin is often the seat of extensive exanthemata and of bloody and encrusted fissures due to scratching. **Enlargement of the spleen** occurs in about one-tenth of all cases.

Not rarely **abdominal dropsy—ascites**—develops, and this may be due to various causes. At times it is dependent upon the general marasmus, and then there will also be cachectic edema of the skin of the lower extremities. In other instances the ascites is due rather to local causes and is unattended with edema of the extremities. It may result from thrombosis of the portal vein, and from obstruction of the portal vein due to pressure exerted by enlarged carcinomatous lymph-glands in the transverse fissure. **Carcinomatous peritonitis**, as a rule, gives rise to the same symptoms as ascites. It results from carcinomatous inoculation of the peritoneum, and causes an accumulation of either serous or hemorrhagic fluid, usually freely movable in the peritoneal cavity. Occasionally excessive tenderness of the abdomen on manipulation is indicative of the presence of peritonitis. Whether the condition be one of ascites or peritonitis, in either instance the presence of a large accumulation of fluid in the abdominal cavity may render examination of the liver extremely difficult and even impossible until the fluid has been withdrawn by puncture.

Individuals suffering from carcinoma of the liver complain principally of a **sense of tension** and a **painful feeling in the right hypochondrium**. Severe pain in the liver may also be present, such as is peculiar to almost all carcinomata, and is prone to occur particularly at night. This may depend upon irritation of adjacent nerves. The **appetite** is generally wanting entirely; the **tongue** is usually **coated**; often the **breath** is fetid. The patients frequently exhibit **eructation** and **vomiting** in the absence of primary carcinoma of the stomach, and this may be attributed to the pressure exerted by the enlarged liver upon the stomach. In addition, in consequence of the cachexia the gastric juice is almost entirely deprived of its free hydrochloric acid, while the absorption-period of the stomach is prolonged and its motor activity is diminished. Occasionally the **supraclavicular lymph-glands** upon the left side are enlarged and indurated, and sometimes also the inguinal glands. This condition may be associated with primary carcinoma of the stomach, although I have observed glandular enlargement in connection with primary carcinoma of the liver also. As a rule, carcinoma of the liver is unattended with **alteration in temperature**. Nevertheless, there may be periods with elevation of temperature, and this may even assume a pronounced intermittent type.

Definite statements as to the *duration of carcinoma of the liver* can hardly be made, as the disease begins most insidiously, and the time of its inception can scarcely be determined. In general a year may be stated to be the maximum duration, although death may occur much earlier from a rapidly growing neoplasm. Even when the disease sets in suddenly with jaundice it should not be forgotten that the jaundice has probably been preceded for some time by the development of carcinomatous tumors. Death may occur under most varied conditions. Often it is the result of *progressive exhaustion*. Occasionally evidences of *blood-dissolution* appear—hemorrhages into the skin and the mucous membranes, particularly also hemorrhage from the stomach (coffee-ground vomiting), in the absence of carcinoma of the stomach, and these may hasten the exhaustion. Occasionally the patients die rapidly in *coma* from *carcinomatous intoxication*, which may be dependent upon auto-intoxication. Excessive accumulation of fluid in the abdominal cavity may cause *death by suffocation*, but at the present day this can be averted by abdominal puncture—a less common occurrence is *rupture of a carcinomatous nodule* through the abdominal wall or into the abdominal cavity, or into an adjacent organ, with its often fatal consequences. Of less importance is extension of carcinoma of the liver along the round ligament to the umbilicus, with the development of carcinoma and carcinomatous degeneration in this situation. It may be quite difficult to discover the primary focus of disease. In any event a decision as to primary carcinoma of the liver should not be reached too hastily, but stomach, rectum, uterus, esophagus, and mammary gland particularly should be carefully examined for a primary focus. Primary carcinoma of the stomach is often beyond recognition in the presence of carcinoma of the liver, because the enlarged liver conceals the stomach and removes it from immediate examination, and the disturbances of gastric activity associated with carcinoma of the stomach (deficiency of hydrochloric acid in the gastric juice, delayed absorption, enfeebled motor activity) may also be present in the absence of anatomic alteration in the stomach solely in consequence of the cachexia induced by carcinoma of the liver. I have even observed coffee-ground vomiting in several cases of carcinoma of the liver, while at the autopsy the stomach was found to be free from malignant disease.

Prognosis.—The prognosis of carcinoma of the liver, like that of carcinoma elsewhere, is unfavorable. Although of late attempts have been made to remove new-growths of the liver by surgical means, but little good can be hoped for from operation in cases of carcinoma of the liver, because the lesion is almost always a secondary one, and only rarely are the conditions so favorable as to permit removal also of the primary neoplasm by operative measures.

Treatment.—No specific remedy for the relief of carcinoma of the liver is known. Symptomatic treatment will include regulation of the **diet**, fats in particular being interdicted in the presence of jaundice. In other respects the treatment will depend upon particularly prominent symptoms that may be present.

Among the remaining tumors of the liver, only *sarcoma* and *adenoma* are of clinical interest. Both varieties of new-growth give rise to the same symptoms as carcinoma of the liver, and are susceptible of differentiation only at autopsy, and at times only after microscopic examination of the tissue. Sarcoma of the liver, like carcinoma, is usually of secondary origin. Melanotic sarcomata occur at times in the sequence of sarcoma of the choroid, even though the diseased eye have been removed many years previously. Occasionally they give rise to melanuria, which is characterized by a brownish or blackish color of the urine when it is evacuated or after standing for a time, or on addition of oxidizing substances (chromic acid, dilute sulphuric acid, boiling with hydrochloric, sulphuric, or nitric acid).

ECHINOCOCCUS OF THE LIVER.

Etiology.—Echinococcus-cysts in the liver represent the **measles of the tapeworm of the dog**—*Tænia echinococcus.* Echinococci are most readily acquired by those who come into frequent and intimate relation with dogs. Echinococcus is most prevalent in Iceland, where every seventh inhabitant is said to be the victim of the disease. Upon the Continent of Europe Pomerania and Mecklenburg are particularly notorious for the frequency with which echinococcus occurs among their inhabitants. The danger of infection is particularly great when the vicious practice of kissing dogs is indulged in, or these animals are permitted to lick the hands or the face, for it is well known that dogs frequently lick the anus, so that it may readily happen that the ova of the Tænia echinococcus remain adherent to the snout, and they may, in the act of licking and kissing, become deposited upon the lips or gain entrance into the mouth of human beings. If, subsequently, with the ingestion of food echinococcus-eggs are swallowed by human beings, the ova, by means of their hooklets, and probably by way of the gastric veins and the portal vein, pass from the stomach into the liver, where they develop into an echinococcus-cyst. In some instances they penetrate also into other viscera, although the liver is the organ most commonly invaded by echinococci. Another mode of infection for human beings consists in the ingestion of echinococcus-ova with inadequately cleansed **vegetables** that have been contaminated by the feces of dogs containing such ova. Contamination of water also with the feces of infective dogs, and infection through the use of such water, may likewise take place. The especial frequency with which echinococcus-disease occurs in Iceland is due to the large number of dogs that are kept, to the intimate relation between the inhabitants and domestic animals, and to a lack of domestic cleanliness.

Anatomic Alterations.—Echinococcus of the liver occurs in two forms—as a *unilocular* and as a *multilocular echinococcus-cyst.* The latter is an exceedingly rare condition that occurs with comparative frequency in Switzerland and in Wurtemburg. *Unilocular echinococcus of the liver* consists of a vesicle with a whitish, opaque, milk-glass-like wall, whose size may attain that of a man's head. It is usually possible to enucleate the cyst from the liver, leaving behind a *connective-tissue capsule* that has resulted in consequence of interstitial inflammation of the liver from the irritation exercised by the echinococcus-cyst upon the surrounding tissue. Incision of the echinococcus-cyst generally gives exit to a *clear fluid.* This resembles water, contains no albumin, but frequently succinic acid, and consequently on addition of a

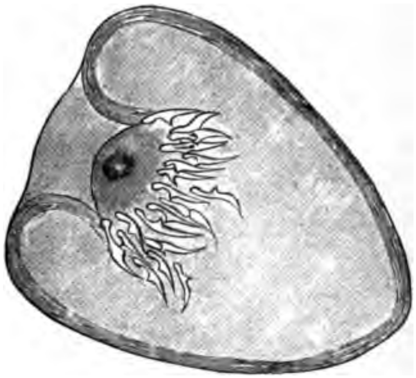

FIG. 54.—Echinococcus-head, with retracted rostellum and crown of hooklets; magnified 275 times (personal observation, Zurich clinic).

dilute solution of ferric chlorid assumes a brownish color. Not rarely small *daughter-cysts* are present in the fluid, and even in these *granddaughter-cysts* and *great-granddaughter-cysts*, which may exhibit much the same peculiarities as the mother-cyst. It is characteristic of the *echinococcus-membrane* that its cut margins become inverted. It may be readily recognized on microscopic examination from the fact that on transverse section it exhibits parallel lines or layers, which have been appropriately compared with the leaves of a book. Upon the inner surface of echinococcus-cysts a glandular mass, the so-called **germinal layer**, is found as a rule. Upon this are situated brood-capsules with echinococcus-heads (**scolices**), which may be readily recognized from the presence of a rostellum and a crown of hooklets and four suckers (Fig. 54). The hooklets resist destructive influences for a long time, so that they are of great diagnostic significance.

At times echinococcus-cysts occur without scolices—so-called *sterile echinococcus-cysts* or *acephalocysts*. Echinococcus-cysts may undergo suppuration. Often their contents are converted into a viscid, *putty-like mass*, in which echinococcus-hooklets are demonstrable.

Although most commonly but one echinococcus-cyst is present in the liver, exceptions to this rule have been observed. Echinococci are most frequently seated in the right lobe of the liver. At times other viscera also, in addition to the liver, are the seat of echinococcus-cysts. *Multilocular echinococcus-cysts* convert the liver into a multinodular, hard organ, which upon section exhibits numerous cysts or chambers filled with softened, pus-like, gelatinous masses. Such alterations were formerly considered as due to soft carcinomata, from which, however, they can be distinguished with certainty by the discovery in them of echinococcus-hooklets on microscopic examination. The condition is dependent upon the development of echinococci within previously formed vessels, most commonly the biliary passages and less commonly the portal vein or the lymphatics, and with the further development continually of new daughter-cysts side by side.

Symptoms and Diagnosis.—Patients with *unilocular echinococcus of the liver* complain frequently at first of a disagreeable sense of **tension** and of **fulness in the right hypochondrium**, which may progress to marked pain. They experience a sense of constriction, and suffer from dyspnea and palpitation of the heart. On examination, the **liver** is usually found **enlarged**, and its limits may be extended both above and below. It is, however, of particular importance to demonstrate the presence of a **fluctuating prominence** upon the surface of the enlarged organ, which corresponds with the echinococcus-cyst. This naturally undergoes respiratory displacement with the liver. It is highly significant if the prominence yields **hydatid fremitus** on percussion. This consists in short-waved vibration, such as is yielded when a wire spring is tapped. The manifestation is, however, by no means frequently present. It is most distinctly appreciable if the percussion-hammer is permitted to rest upon the prominence for a few seconds following each blow, or if the middle three fingers of the left hand are placed upon the prominence, and the central one of these is percussed with a hammer or with the middle finger of the right hand. In rare instances the hydatid fremitus is associated with a deep sonorous note on auscultation.

In the *differential diagnosis* from other fluctuating prominences upon the surface of the liver, soft carcinomata and abscess of the liver particularly should be borne in mind. Carcinoma of the liver, however, occurs only in advanced life, is attended with rapid emaciation, and is usually associated with the presence of primary carcinoma in some other viscus: while abscess of the liver is attended with fever, and the etiologic factors (gall-stones, ulceration in the intestine and the genito-urinary apparatus, septicopyemia) should be taken into consideration. *Exploratory puncture* is an extreme means of differentiation, yielding in the case of echinococcus of the liver a clear, colorless, watery fluid, containing no albumin, but hooklets, and possibly even scolices and succinic acid. Whenever possible, exploratory puncture should be avoided, as the contents of the cyst may readily leak into the abdominal cavity through the orifice of puncture, so that septic peritonitis may result, or, what is less dangerous, symptoms of auto-intoxication may appear in the form of widespread urticaria. At times sudden death has followed exploratory puncture, and this likewise has been attributed to auto-intoxication.

Naturally, it will be possible to feel fluctuating prominences distinctly upon the surface of the liver only when the echinococcus-cyst is applied directly to the abdominal wall. The conditions are less simple when the tumor is covered by the thoracic wall, although it may be that the cyst is applied to the wall with such pressure as to cause enlargement of some intercostal spaces, while those above and below are crowded together, and a visible projection forms externally, which may yield fluctuation and hydatid fremitus. Occasionally echinococcus-cysts are situated in the neighborhood of the upper border of the liver, and are thus inaccessible to the eye and the hand. They can then be demonstrated only by careful determination of the upper border of the liver, which suddenly deviates from its horizontal course, and makes a marked, usually hemispherical deflection upward into the thoracic cavity. In contradistinction from encapsulated pleural exudates and subphrenic collections of pus, this projection will exhibit respiratory displacement. Here also exploratory puncture would be decisive in doubtful cases. All other manifestations attending echinococcus of the liver are rather of accidental and therefore of subordinate importance. Among these may be included *jaundice*, which develops when pressure is exerted upon the larger biliary passages; *ascites*, in consequence of pressure upon the trunk of the portal vein; and *gastro-intestinal disturbances*, as a result of the crowding of the abdominal contents.

The *danger from a unilocular echinococcus-cyst of the liver* depends principally upon its *tendency to continued growth.* In consequence the diaphragm, the lungs, and the heart may be so greatly displaced upward that *death* will result *from suffocation.* Not rarely *rupture of the echinococcus-cyst* occurs *into adjacent organs.* It may take place into the stomach, as will be indicated by the vomiting of echinococcus-vesicles. Should rupture occur into the intestine, the urinary passages, or, after previous adhesion between the lungs and the diaphragm, into the bronchial tubes, echinococcus-vesicles will appear in the stools, the urine, or the expectoration respectively. The expectoration under such conditions acquires a penetrating, aromatic odor, suggestive of prune-juice. At times it is also characterized by a red color, in consequence of the abundant presence of hematoidin-crystals. Rupture into the peritoneum, the pleura, or the pericardium will be followed by peritonitis, pleuritis, or pericarditis. Rarely rupture takes place into the hepatic veins or the inferior vena cava. Under such conditions echinococcus-vesicles may gain entrance into the right heart and the pulmonary artery, and cause sudden death from embolism of this artery. Rupture through the abdominal walls has also been observed. Should *suppuration take place in an echinococcus-cyst,* symptoms will appear similar to those of abscess of the liver, and often the nature of this collection of pus will be explained only by the discovery in it of echinococcus-hooklets. Echinococcus-cysts situated not upon the surface of, but within, the liver may be inaccessible to diagnosis during life. Under favorable conditions a probable diagnosis might be made in the presence of enlargement of the liver for which no other cause can be ascertained. The *course of a unilocular echinococcus-cyst of the liver* is usually chronic, and may extend over several years.

Multilocular echinococcus of the liver is generally attended with profound jaundice and enlargement of the spleen. The liver itself is enlarged, and feels unusually hard and nodular. The disease may readily be confounded especially with carcinoma and with hypertrophic cirrhosis of the liver. Carcinoma of the liver, however, usually develops late in life, and in association with primary carcinoma in some other viscus; and hypertrophic cirrhosis of the liver with jaundice is generally attended with increase in the size of the liver, with preservation of the smoothness of its surface. Death occurs generally with progressive exhaustion, but at times only after the lapse of more than ten years.

Prognosis.—The prognosis of echinococcus of the liver is under all conditions serious. It has recently been rendered less unfavorable for unilocular cysts, from the fact that cysts have been successfully removed by operative intervention.

Treatment.—**Prophylactic measures** include domestic cleanliness, treatment directed to the expulsion of the Tænia echinococcus in dogs, and the avoidance of licking by dogs. The kissing of dogs may be followed by most serious dangers. If echinococcus-cysts have developed in the liver, they should be removed by **operation.** Recently atrophy of the cysts has been induced by **injections of mercuric chlorid.** Internal remedies (potassium iodid, ether) and affusions of a solution of sodium chlorid have been unattended with success. In the presence of a *multilocular echinococcus* of the liver, there is scarcely any other remedy than the relief of especially threatening or troublesome symptoms by the usual measures. Further, a large portion of diseased liver has in one instance been successfully removed by operative means.

DISPLACEMENTS OF THE LIVER (HEPATOPTOSIS).

In women who have borne children the liver is not rarely found displaced downward and unduly mobile. Obviously relaxation of the suspensory ligaments of the liver and of the abdominal walls is the cause of this condition. *Enlargement of the liver*, as a result of stasis, of fatty or amyloid degeneration, of the presence of parasites or of neoplasms, is also capable of causing downward displacement of the liver in consequence of the increased weight of the organ. At times, however, the condition appears to be dependent upon *congenital weakness of the suspensory ligaments of the liver*, or such weakness may be acquired as a result of *severe or protracted disease. Nervous influences* also are of significance. Bodily over-exertion, expulsive efforts, and constriction by clothing favor the development of the condition. The degree of displacement of the liver is susceptible of great variation. In aggravated cases the liver may be found in the pelvis, and in place of the normal hepatic dulness at the base of the right chest anteriorly a tympanitic percussion-note is obtained from the presence of intestine, and this will disappear when the liver is replaced in its normal situation. Such a condition is designated as *movable* or *wandering liver*. Further, in forming an opinion as to the identity of a foreign body in the abdominal cavity from its shape great care must be observed, as errors have been made in mistaking a movable body in the abdomen for a wandering liver, while the autopsy has disclosed carcinoma of the omentum. It is especially important if one or two incisures (for the gall-bladder and the round ligament) are found in the lower border of the liver. At times the stretched round ligament also may be palpable through the abdominal wall. Occasionally wandering liver is detected accidentally upon examination of the abdomen. If the patient on palpating the abdomen has, perhaps, accidentally discovered the presence of a movable body, he is often so greatly distressed by the fear that the condition is due to a malignant neoplasm that a positive and reassuring opinion on the part of the physician will be required to convince the patient that the state of affairs is not serious. Some patients complain of a disagreeable, at times also painful, sense of traction and tension in the abdomen, and gradually become nervous and hysterical. Derangement of intestinal activity also may develop if loops of bowel are compressed or distorted.

The **prognosis** is favorable with regard to life if the condition is not dependent upon tumors or parasites, although it is often impossible to effect a permanent cure.

The **treatment** must generally be mechanical, attempts being made by means of **abdominal bandages** applied to the lower half of the abdomen to exert pressure upward, and thus to force the liver into its normal situation. **Massage of the abdomen** also appears worthy of recommendation. Further, attention should be given to the general condition.

Congenital displacement of the liver may occur in association with transposition of the viscera. Under such conditions the liver will be situated in the left, and the spleen in the right concavity of the diaphragm. The displacement may involve only the liver and the spleen. As a rule, however, the remaining organs of the thoracic and abdominal cavities are correspondingly displaced. The condition is unattended with discomfort or danger.

CONSTRICTED OR FISSURED LIVER.

Constricted or fissured liver may result from tight lacing. The fissure formed is generally horizontal, and within it the serosa covering the liver is thickened, tendinous, and opaque. Beneath the fissure the liver-tissue may be entirely wanting, so that a portion of the liver may be completely constricted off. Often the latter is thickened and exhibits signs of blood-stasis. Most commonly the lowermost portion of the right lobe of the liver is constricted off, although at times the fissure may extend without interruption into the left lobe. Often the condition remains latent throughout life if the lower border of the liver does not extend considerably below the costal margin. Under other conditions a horizontal depression may be palpable upon the lower portion of the surface of the liver beneath the abdominal wall. Below this depression the constricted portion of liver appears thickened, and at times can be reflected upward. Care must be taken to avoid confusion especially with a syphilitic lobulated liver. The condition is unattended with danger. Interna ment is unnecessary, and would also yield no result. Of late the constricted portion of the liver has been removed by operation in a number of instances.

DISEASES OF THE BILIARY PASSAGES.

CATARRH OF THE BILIARY PASSAGES (CATARRHAL CHOLANGITIS AND CHOLECYSTITIS).

Etiology.—Catarrh of the biliary passages is an exceedingly common disorder, and is induced particularly by **infectious or toxic influences.** Most commonly it is secondary to **catarrh of the gastroduodenal mucous membrane,** the inflammatory process extending directly from the mucous membrane of the duodenum to that of the choledoch duct, and the more remote biliary passages. At times catarrh of the biliary passages arises in the sequence of **acute or chronic infectious diseases,** as, for instance, fibrinous pneumonia, typhoid fever, erysipelas, pharyngeal diphtheria, and syphilis. Occasionally catarrh of the biliary passages occurs as an **independent infectious disease.** Of this character were especially certain epidemics of jaundice that have at times been observed in over-crowded dwellings (barracks, prisons), in the sequence of revaccination and the ingestion of spoiled food and drink. A number of such epidemics have occurred particularly in the autumn months. At times children were attacked in especially large numbers. Among the *toxic catarrhal states of the biliary passages,* that following phosphorus-poisoning should be mentioned particularly. This may, however, leave the larger biliary passages intact, and attack only the smaller intrahepatic biliary passages. The *hypostatic catarrh of the biliary passages* that occurs particularly in association with chronic disease of the heart and the respiratory organs is worthy of mention. Whether circulatory and catarrhal alterations in the biliary passages are operative in connection with the jaundice that appears in some women shortly before and during the menstrual period—so-called *menstrual icterus*—is as yet unexplained. Not rarely **gall-stones** cause catarrh of the biliary passages. Obviously the mechanical irritation of the mucous membrane of the biliary passages by the calculi is particularly favorable to the deposition of microörganisms, most commonly the Bacterium coli, upon the mucous membrane, with the development of an inflammatory process. Not rarely **diseases of the liver** are attended with catarrh of the biliary passages, often because by causing obstruction of the biliary passages they give rise to biliary stasis, and the latter may be followed by inflammation, as under such circumstances bacteria readily gain entrance into the biliary passages, particularly the Bacterium coli.

Anatomic Alterations.—In the presence of catarrh in the large biliary ducts the discharge of bile is not rarely prevented by a *plug of mucus.* At times, however, the plug has been forced into the intestine, so that its previous existence can only be recognized from the fact that the biliary passages are discolored only up to the point previously occupied by the plug, while they are unstained for the remaining distance to the intestine. The mucous membrane is often conspicuous by reason of swelling and marked secretion of mucus. Stenosis of the biliary passages has given rise to biliary stasis, and thereby to the icteric manifestations in the liver and the remaining viscera that have been described on pp. 321 and 322. Catarrh of the intrahepatic biliary passages can be demonstrated in the smaller biliary channels only with the aid of the microscope, active desquamation of the epithelial cells particularly being found.

Symptoms, Diagnosis, and Prognosis.—Catarrh of the biliary passages can be recognized only when in consequence of the formation of a plug of mucus and tumefaction of the mucous membrane, **biliary stasis** and **hypostatic and resorptive icterus** have developed, and which, from the nature of the causative factors, is also designated *catarrhal jaundice.* The symptoms are identically those that have been described on pp. 317–321 in the general consideration of jaundice, and in the diagnosis the essential point is to determine whether catarrh of the biliary passages is responsible for the jaundice. In this connection the history and the results of physical examination of the viscera are of great importance. Often previous dietetic error, nausea, vomiting, constipation, or diarrhea will leave no room for doubt that catarrh of the biliary passages has been preceded by gastroduodenal catarrh, and the condition is then designated *gastroduodenal jaundice.* When primary infectious catarrh of the biliary passages is present the pronounced involvement of the general condition is often striking. The liver and particularly the spleen are considerably enlarged. It is just in this variety of catarrh of the biliary passages that cholemia may readily develop, and death take place. The *duration* and the *course of catarrhal jaundice* depend upon the causative factors. Recovery from gastroduodenal jaundice usually takes place within one or two weeks, while the jaundice accompanying incurable diseases of the liver persists until death. Also the *prognosis* depends naturally upon the character of the causative factors.

Treatment.—In the first place, on account of the existing biliary stasis and jaundice the **diet** and the **state of the bowels** should be regulated in accordance with the rules laid down on pp. 322 and 323. Should the disorder be secondary to gastro-intestinal catarrh, the employment of **acids** is advisable. Attempts have been made, further, to cause expulsion from the biliary passages of possible accumulations of mucus by **pressure with the fingers upon the dilated gall-bladder** not rarely distended by accumulated bile, or by inducing contraction by means of the **faradic current.** It has been stated that as a result of this manipulation a gurgling murmur is appreciated, after which the biliary passages have become again patulous. Particularly troublesome symptoms of jaundice will require the treatment described on pp. 322 and 323.

PURULENT INFLAMMATION OF THE BILIARY PASSAGES (PURULENT CHOLANGITIS AND CHOLECYSTITIS).

Purulent inflammation of the biliary passages scarcely occurs apart from the influence of *bacteria*, and according to previous experience particularly of the Bacterium coli commune, the Streptococcus pyogenes, the Staphylococcus pyogenes albus and aureus, the pneumoniacoccus, and the typhoid-bacillus. The condition is usually a secondary disorder, occurring particularly in connection with the presence of *gall-stones* or *parasites in the biliary passages*, and with *infectious diseases*, as, for instance, in the sequence of typhoid fever, and occasionally also after *abscess of the liver* and *pylephlebitis*. *Anatomically* the disorder is characterized by inflammation of the mucous membrane of the biliary passages, with the presence of pus. Occasionally the gall-bladder particularly is filled with pus, and distended thereby, the condition being designated *empyema of the gall-bladder*. The disease is exceedingly difficult of recognition. Particular diagnostic significance should be attached to the *etiologic factors*. In addition there will be the clinical picture of general *septicopyemia* (fever, enlargement of the spleen, metastatic inflammation in other organs, occasionally ulcerative endocarditis). Of importance naturally are *local alterations in the liver and the biliary passages*, among which enlargement of the liver, jaundice, tenderness, and, in the presence of empyema of the gall-bladder, the demonstration of the enlarged gall-bladder, with tenderness on pressure, may be mentioned.

The **prognosis** is grave, as death may result from septicopyemia, or metastatic suppuration, or rupture of the biliary ducts into the liver, with the formation of a liver-abscess; into the portal vein or the hepatic veins; into the abdominal cavity, with rapidly fatal peritonitis; or into some other internal organ; or through the abdominal wall in the presence of empyema of the gall-bladder. In the last-named event a *biliary fistula* may develop, from which occasionally numerous gall-stones are evacuated for a long time.

The **treatment** *of empyema of the gall-bladder* is surgical. The gall-bladder should be opened—**cholecystotomy**—and pus and gall-stones evacuated; or it should perhaps be entirely removed—**cholecystectomy**—the latter particularly when the wall of the gall-bladder is the seat of profound ulceration and alteration. Should the suppurative inflammation involve rather the biliary passages, it may also become necessary to expose these, to incise them, and evacuate the calculi—**choledochotomy**; otherwise the only other remedy available would be to sustain the strength of the patient as well as possible by means of the administration of large amounts of **alcohol**, and to employ **disinfectants**, among which sodium salicylate (1.0—15 grains—every two hours) and salol (1.0—15 grains—every two hours) may be recommended. In addition a hot cataplasm should be applied over the liver.

DROPSY OF THE GALL-BLADDER.

The conditions for the development of dropsy of the gall-bladder are provided whenever *permanent occlusion of the cystic duct* has been brought about. This occurs most commonly in the presence of gall-stones, either because a gall-stone has become impacted in the cystic duct or because in its progress it has injured the wall of the duct, so that subsequently adhesions form at the site of injury. Under such conditions the bile is gradually absorbed from the obstructed gall-bladder, while a colorless mucoid fluid takes its place. The amount of fluid may reach 40 quarts, so that the gall-bladder becomes correspondingly enlarged.

Among the most important **symptoms** of dropsy of the gall-bladder is the presence of **enlargement of the gall-bladder.** The gall-bladder may be visible to the eye when the abdominal wall is thin, and under other circumstances it may be palpable as a tensely distended, smooth, pear-shaped tumor. From a diagnostic point of view it should be borne in mind that the tumor undergoes respiratory displacement with the liver, is situated at the lower border of the liver, and that above it an incisure may be recognized, and that the dulness to which it gives rise passes over immediately into that of the liver. At times I have observed diagnostic difficulties to arise from the lodgment of the transverse colon between the lower border of the liver and the lower extremity of the gall-bladder, so that the tumor to which the latter gave rise was separated from the hepatic dulness by a tympanitic zone. Further, the tumor had the shape of the kidney, and was readily movable in the abdominal cavity. Slight enlargement of the gall-bladder scarcely occasions discomfort, and at most may cause alarm in a patient who accidentally discovers the presence of a tumor in the abdominal cavity, and considers it carcinomatous. Excessive dropsy of the gall-bladder causes a sense of tension and pain in the region of the liver, and by displacement of the contents of the abdominal cavity may give rise not only to gastro-intestinal disturbances, but, by displacement of the diaphragm upward, also to dyspnea, palpitation of the heart, and danger of suffocation.

Only surgical **treatment** is available. **Puncture of the gall-bladder** is not free from danger, because the contents of the viscus may escape into the abdominal cavity and give rise to fatal peritonitis. **Incision of the gall-bladder—cholecystotomy**—is attended with the possibility that fluid may reaccumulate in the gall-bladder. Therefore recovery can be brought about most certainly by extirpation of the gall-bladder—**cholecystectomy**.

CARCINOMA OF THE BILIARY PASSAGES.

Carcinoma of the biliary passages is situated most commonly at the mouth of the choledoch duct or in the gall-bladder itself. *Carcinoma at the mouth of the choledoch duct* gives rise to *chronic jaundice*, which usually terminates fatally with progressive exhaustion, and the cause of which is, as a rule, not recognized during life. *Carcinoma of the gall-bladder* gives rise to the development of a nodular, usually painful, and frequently extensive tumor, situated in the region of the gall-bladder, usually appearing as a pear-shaped swelling, and undergoing respiratory displacement with the liver. The percussion-dulness of the liver and that of the tumor are continuous. Frequently, but by no means constantly, jaundice is present. Often the new-growth extends to the liver, and a secondary neoplasm develops in this organ, which may attract the greater amount of attention. Death will probably occur within a year amid excessive asthenia. Like carcinoma in other organs, similar disease of the biliary passages usually occurs after the fortieth year of life. The causative factors remain, as a rule, unrecognized. The frequent association with gall-stones suggests that mechanical irritation may excite the development of carcinoma. Nevertheless, the gall-stones may also have developed secondarily in consequence of biliary stasis. The significance of the gall-stones appears naturally to be supported by the fact that in cases of secondary carcinoma of the gall-bladder calculi do not occur in this viscus.

The **prognosis** is unfavorable, as with carcinoma in other situations. Recently carcinoma of the gall-bladder has been successfully removed with the knife in a number of cases. Beyond such measures, *symptomatic treatment* alone will be applicable.

PARASITES IN THE BILIARY PASSAGES.

Parasites in the biliary passages are rare. *Echinococci* have already been mentioned in the consideration of multilocular echinococci of the liver. *Flukes—Distoma hepaticum* and *Distoma lanceolatum*—may cause jaundice, purulent inflammation of the biliary passages, and suppuration in the liver, and are susceptible of recognition only when parasites or their ova are present in the stools.

At times a *spool-worm—Ascaris lumbricoides*—may wander from the intestine into the biliary passages and cause obstruction and jaundice, and at times suppuration in the liver or the formation of gall-stones.

GALL-STONES (CHOLELITHIASIS).

Etiology.—Extremely little of a definite nature is known as to the causes of gall-stones. Experience has shown that all those conditions favor the formation of gall-stones that cause *biliary stasis.* These include a **sedentary mode of life** and **constricting clothing.** Thus, gall-stones have been frequently found in association with constricted liver. Further, they occur about four times as frequently in *women* as in men. What influence food may exert is unknown. If *obesity* predisposes to the formation of gall-stones, it is possible that biliary stasis is principally also the actual etiologic factor, as a result of the crowding in the abdominal cavity in consequence of the excessive fat in the omentum and the mesentery. Why gall-stones should be more common in certain *regions* than in others is as yet unexplained. Children but rarely suffer from gall-stones, which generally develop after the thirtieth year of life.

Anatomic Alterations.—Gall-stones develop most commonly in the *gall-bladder*, and much less commonly in the intrahepatic biliary passages. Gall-stones in the hepatic duct, the cystic duct, or the choledoch duct have always migrated from the gall-bladder or the liver. Gall-stones consist either of *bilirubin and lime-salts* or of *cholesterin*, or they may be of *mixed composition*, either layers of bilirubin and lime-salts and cholesterin alternating with each other on cut section, or the one constituting the nucleus or the outer layer of the calculus. Bilirubin-calculi are brownish in color, while cholesterin-calculi are white, upon section present a crystalline appearance with a radiate arrangement, and yield a greasy sensation to touch.

Rarely, biliary calculi consist of *calcium carbonate.* They are whitish in color, and are characterized by great hardness.

According to the *size of biliary precipitates*, a distinction is made between *biliary sand* and *biliary calculi.* The former possesses the character of sand and may attain the size of a pin-head. The size of gall-stones depends principally upon their number, for the more numerous the stones the smaller must they be. At times the entire gall-bladder is occupied by a single gall-stone, measuring several centimeters in length and reproducing the pear-shape of the gall-bladder. At times two or more gall-stones are present [illegible] bladder, and these may be in contact by a sort of [illegible] *of gall-stones* may reach many hundreds, and even thousands. Often, gall-stones that lie side by side in considerable number acquire peculiar shapes. They have been designated *facetted gall-stones*, and have been appropriately compared with the shape of the carpal bones. These peculiarities are not dependent, as has been supposed by some, upon the mutual attrition of adjacent angles and surfaces, but upon a failure in development at such points as contiguous stones, which originally possess a roundish shape, lie in contact. Such gall-stones generally exhibit extremely smooth surfaces, as if polished. In contradistinction from these there occur also globular calculi, with a multinodular surface, and these have been designated *mulberry-calculi*.

Opinions are divided as to the *mode of origin of gall-stones*. In any event, the view is to be rejected that bilirubin or cholesterin is present in the bile in such excessive amount as not to be retained in solution, but to be precipitated. As experience has shown that *biliary stasis* favors the formation of gall-stones, there is much in favor of the view that the primary impulse in the formation of gall-stones is to be referred to *bacteria*, which, in the presence of biliary stasis, may readily gain entrance from the intestine into the biliary passages. In the first place, the Bacterium coli commune will suggest itself, because it is a constant inhabitant of the intestine. The bacteria excite catarrhal inflammation of the mucous membrane of the biliary passages, and for this reason some clinicians have employed the designation *calculus-forming catarrh*. The inflammatory products exert a decomposing influence upon the biliary acids, and as the biliary salts retain bilirubin and cholesterin in solution in the bile, these substances are precipitated when decomposition of the biliary salts takes place. The precipitated constituents of the gall-stones do not simply crystallize together, but are held together by an organic mass. This material can be demonstrated when gall-stones are carefully dissolved. In some instances gall-stones have been found to develop around *foreign bodies* (prune-stones, blood-clots, ascarides).

Symptoms and Diagnosis.—Many more persons suffer from gall-stones than is generally believed, because often the calculi are *latent* and occasion no symptoms. At times gall-stones are discovered accidentally, the *gall-bladder*, upon examination for some other cause, being found to be not only enlarged, but also filled with gall-stones. The latter will be recognized as hard, polyhedral bodies, which not rarely can be rubbed against one another with a palpable crackling sound, and yield the same sensation as when a bag filled with small pebbles or nuts is manipulated between the fingers. In cases in which the diagnosis was doubtful a sterilized needle has even been introduced into the gall-bladder, and observation made whether a hard body was encountered with the point of the instrument.

Rarely, the presence of gall-stones is disclosed by their passage through the biliary ducts without inducing symptoms, and their discovery *accidentally in the stools*. Gall-stones often give rise to **hepatic colic**. This results from the escape of the calculi from the gall-bladder, and their passage through the biliary ducts into the intestine. Under such conditions they readily become impacted in the biliary ducts, causing biliary stasis and pain, and giving rise to the clinical picture of hepatic colic. Calculi from the intrahepatic biliary ducts do not give rise to hepatic colic in their passage into the intestine, as they traverse channels that in comparison with their site of origin become progressively larger as the intestine is approached. Frequently no especial causes for the occurrence of biliary colic are demonstrable, while in some instances anger and other emotional disturbances, bodily concussion, or physical over-exertion has been an antecedent condition. Menstruation, pregnancy, and the puerperium without doubt also favor the occurrence of attacks of hepatic colic.

The principal symptom of biliary colic is **pain.** This is at times confined strictly to the region of the gall-bladder (external border of the right abdominal rectus muscle, just below the right costal margin), although it often radiates throughout a larger area, particularly toward the epigastrium, the right scapular region, and often also to the right arm. The pain increases paroxysmally to an unbearable degree, so that the patients cry aloud and groan, exhibit a painful and anxious expression, and are covered with perspiration. Occasionally, there may be loss of consciousness and general clonic muscular spasm, obviously because the pain causes reflex spasm of the cerebral arteries, and thereby cerebral anemia. A **chill** likewise is not rarely induced by reflex influences, and this may be repeated several times. Vomiting also is a frequent symptom dependent upon reflex influences. Often **elevation of temperature** takes place, although I would not attribute this to nervous influences, but to inflammation of the biliary passages, and would consider it an indication of septic general infection.

The occurrence of **jaundice** is of great importance, developing on the average in the course of three days, as this length of time is necessary for the blood to become sufficiently saturated with biliary coloring-matter in order to stain the skin and the mucous membranes yellow. The degree of jaundice varies extremely; at times there is but slight yellowish discoloration of the conjunctivæ. The jaundice naturally persists for some time after the attack of hepatic colic, until the biliary coloring-matter has been removed (principally through the urine).

It is noteworthy that when the choledoch duct is obstructed by a gall-stone *enlargement of the gall-bladder* in consequence of biliary stasis takes place but seldom.

The *duration of an attack of hepatic colic* is susceptible of great variation, and fluctuates between a few hours and as many weeks. In the latter event the patients are never free from pain, although this at times becomes more severe. Under such conditions they not rarely lose strength in an alarming degree. There are also great variations in the *recurrence of attacks of hepatic colic.* One must be prepared for this if gall-stones have not been found in the stools, in spite of the fact that these have been thoroughly dissolved upon a sieve in a stream of water. To obtain assurance upon this point the stools must be continuously examined for at least a week after the pain has disappeared, as biliary calculi may remain for some time in the intestine. Gall-stones naturally will not be found in the stools if they slip back into the gall-bladder. The occurrence of facetted biliary calculi in the stools will justify the anticipation of recurrent attacks of hepatic colic, because such calculi are usually multiple in the gall-bladder. It is, further, noteworthy that biliary calculi in the stools must by no means always have passed through the biliary passages, and it appears to occur not at all rarely that they give rise to inflammation of the biliary passages and adhesions to the bowel, particularly the transverse colon, and then after perforation gain entrance into the bowel through a *biliary-intestinal fistula.* When gall-stones have entered the bowel the danger is not always passed, for occasionally they may occupy the lumen of the intestine in such a peculiar manner, or a number of stones may be joined together by fecal matter into so large a mass, as to cause obstruction of the bowel and the development of ileus.

In rare cases gall-stones in the cecum or the vermiform appendix give rise to *typhlitis, appendicitis, perityphlitis,* or *paratyphlitis.*

Occasionally biliary calculi become so firmly impacted in the cystic duct or the choledoch duct that they cannot be moved either forward or backward. Such *impaction of gall-stones* is attended with the danger of inflammation, pressure-necrosis, and perforation of the gall-stones into the abdominal cavity, and this will generally be followed by rapidly fatal peritonitis. Attacks of hepatic colic are not always attended with such readily recognized manifestations as have been described. Occasionally they give rise to vomiting so severe and persistent as to arouse suspicion of ulceration of the stomach. At times, also, the pain is situated in the epigastric region, and this would appear to strengthen the suspicion of gastric ulcer. In other instances pain in the scapular region, about the right side of the chest, or along the right arm is such a conspicuous feature that gall-stones may be confounded with neuralgia. At times chills recur daily at the same hour, and the clinical picture is suggestive of intermittent fever, although malarial plasmodia are not found in the blood. If the pain radiates actively into the right iliac fossa, care will be necessary to avoid confusion with perityphlitis. In all doubtful cases attention should be directed to the occurrence of jaundice, which naturally often occurs in slight and scarcely appreciable degree.

In addition to hepatic colic and its consequences, biliary calculi not rarely excite *inflammatory alterations in the biliary passages*, suppuration and septicopyemia, and it is often exceedingly difficult to recognize them under such conditions. I have observed *ulcerative endocarditis, otitis media*, and *inflammation of the cerebral sinuses* develop in the sequence of cholecystitis due to gall-stones. Occasionally *abscess of the liver* develops as a result of gall-stones. Also at times the gall-bladder becomes attached by adhesions to adjacent organs (stomach, intestines, urinary passages), ruptures into these, and evacuates its calculous contents into them, so that gall-stones may be vomited or be found in the urinary bladder. Occasionally adhesions to the abdominal wall form, with rupture of the gall-bladder externally, and at times numerous gall-stones are evacuated through the resulting *external bilary fistula* for a considerable length of time. Rupture of gall-stones into the lungs and the bronchi has also been observed. *Pericholecystitis* is a common sequel of gall-stones. *Gall-stones may persist* at times throughout life. Only rarely is the disease terminated with a single attack of hepatic colic.

Prognosis.—Hepatic colic is a serious condition under all circumstances, being attended with numerous dangers, whether the calculi exhibit a migratory or a sessile tendency. It should not be forgotten in this connection, however, that gall-stones may excite the development of *carcimoma* in the gall-bladder. At times, also, connective-tissue hyperplasia develops in the neighborhood of the biliary passages within the liver, and gives rise to *biliary cirrhosis of the liver*.

Treatment.—Patients with gall-stones generally seek professional assistance at the time of an attack of *hepatic colic*, and for the relief of which the following measures may be adopted: Patients should remain in bed and apply a hot **poultice over the region of the liver**. They should partake of **liquids** only, such as weak tea, milk, and wine, diluted with water. If the pain is severe, a **subcutaneous injection of morphin** should be given:

℞ Morphin hydrochlorate, 0.3 (4½ grains),
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: From 0.25 (4 minims) to 0.5 (8 minims) subcutaneously.

The patient should never be entrusted with morphin and a syringe for use upon himself because the danger and the temptation are too great to make injections when not necessary, and gradually to employ larger doses of the drug, and finally to become a morphin-habitué. Daily rectal injections of water at room-temperature may be recommended, as stimulation of intestinal peristalsis appears to favor the passage of the gall-stones through the biliary channels.

Enemata of oil have been advised by some physicians. *Olive-oil* (180.0—6 fluidounces) internally and *glycerin* have been recommended to facilitate the passage of gall-stones and for the relief of attacks of hepatic colic.

When an attack of biliary colic has been successfully relieved, efforts should next be made to effect solution of gall-stones still present, and to prevent the formation of new calculi. **Oil of turpentine** and **ether** in the form of Durande's remedy especially have been recommended as a solvent:

℞ Oil of turpentine,	5.0 (75 minims),
Ether,	20.0 (5 fluidrams).—M.

Dose: 20 drops four times daily.

A solvent action by this mixture can naturally be expected only upon cholesterin-calculi. Nevertheless an undeniable effect upon bilirubin-calculi also is at times observed, but under such conditions the result must be attributed less to a solvent action than to the circumstance that the remedy stimulates the secretion of bile. Sodium salicylate acts in the same way:

℞ Sodium salicylate, 1.0 (15 grains).
Make 10 such powders.
Dose: 1 powder thrice daily after eating.

The administration of **bile** and its **sodium-combination** also favors the secretion of bile, and may be advised in the treatment of gall-stones. Courses of treatment with the **waters** at springs are much employed, particularly at Carlsbad, Marienbad, Tarasp, Kissingen, Homburg, Ems, Wiesbaden, and Vichy. That these waters are capable of exerting a solvent effect has not been demonstrated; they do, however, influence favorably the catarrhal conditions of the gastro-intestinal mucous membrane and the biliary passages, and in this way may prevent the formation of gall-stones. Persons in moderate circumstances may, with the same object in view, use artificial Carlsbad salt for periods of four weeks:

℞ Artificial Carlsbad salt, 100.0 (3 ounces).
Dose: 1 teaspoonful in a pint of tepid water, to be taken in four equal parts at intervals of ten minutes.

Persons suffering from gall-stones should take considerable exercise, not wear constricting clothing, secure a daily evacuation of the bowels, and avoid all conditions capable of causing biliary stasis.

Recently the **operative treatment of gall-stones** has occupied attention. Surgical intervention will be justified in the presence of *impaction of a gall-stone* in a biliary duct. With this end in view the abdomen should be opened, the stone located in the biliary duct, and an attempt made by gentle pressure to force its entrance into the intestine or the gall-bladder, or to crush it between the fingers—*cholelithotripsy*. Otherwise, it would be necessary to incise the bile-duct—*choledochotomy*—extract the stone, and suture the margins of the wound. At times impacted calculi remain *in situ* without exciting inflammation, although the chronic hypostatic jaundice gives rise to alarming emaciation. Under such circumstances *cholecystenterostomy* has been performed; that is, an artificial communication has been established between the gall-bladder and the highest possible loop of small intestine. It may happen, of course, that the biliary passages are infected with bacteria from the intestine, and become inflamed. Should there be a marked tendency to the formation of gall-stones, and should attacks of hepatic colic occur with such frequency as to make the life of the patient miserable, or should empyema of the gall-bladder have developed in consequence of the presence of gall-stones, this viscus has been incised—*cholecystotomy*—or it has been removed—*cholecystectomy*. The latter operation is scarcely more dangerous than the first, and has the further advantage that no new gall-stones can form in the gall-bladder. Under the worst of circumstances the development of gall-stones in dilatations of the large biliary ducts would be possible. When gall-stones have become impacted in the intestine, and symptoms of ileus have developed, abdominal section has been performed, the bowel incised, the calculus extracted, the intestine and the abdomen sutured, and cure effected in this way.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER.

THROMBOSIS OF THE PORTAL VEIN (PYLETHROMBOSIS).

Etiology.—As in other blood-vessels, thrombi may form in the portal vein as a result of **compression**, of **slowing of the blood-stream**, and of **marantic conditions**. *Pressure-thrombosis* is observed in connection with tumors surrounding the trunk of the portal vein, as, for instance, carcinoma of the pancreas, stomach, or intestine, enlargement of the portal lymphatic glands, and at times also as a result of compression due to peritonitic adhesions. *Thrombosis of the portal vein due to slowing of the blood-stream* develops particularly in association with certain diseases of the liver, most commonly chronic interstitial hepatitis and carcinoma of the liver. *Marantic pylethrombosis* is observed finally in the sequence of protracted and debilitating diseases, among which chronic diarrhea, carcinoma, and pulmonary tuberculosis may be mentioned. Thrombosis of the portal vein is a rather uncommon disorder.

Symptoms, Diagnosis, Prognosis, and Anatomic Alterations..—Obstruction of the trunk of the portal vein by a thrombus gives rise necessarily to increase in the blood-pressure in the portal area, and consequently to **portal stasis.** The latter is manifested by the occurrence of *ascites, splenic enlargement, hypostatic catarrh of the stomach and the intestine,* and *hemorrhoids,* at times of *hemorrhage from the mucous membrane of the stomach and the intestine. Jaundice* also may develop, and this has been explained by the fact that in consequence of the thrombosis the blood-pressure in the intrahepatic branches of the portal vein has fallen so low that, contrary to the rule, bile can pass over from the biliary capillaries into the portal branches. It is therefore evident that the same conditions are present as in cirrhosis of the liver, and nothing else would be expected, as in the latter disorder, also, the portal area is partially obstructed and partially constricted, although the process involves not the trunk of the portal vein, but its intrahepatic branches. It has even been maintained that pylethrombosis gives rise to chronic interstitial hepatitis, but this view has not unjustly been contradicted.

The *differential diagnosis* between **cirrhosis of the liver** and pylethrombosis can scarcely ever be made with certainty. Excessive indulgence in alcohol is suggestive of cirrhosis, which, further, is rather a common disorder. Pylethrombosis may also be readily confounded with **chronic-serous, tuberculous,** and **carcinomatous peritonitis,** and in the differentiation the points mentioned on page 336 should be borne in mind.

The *prognosis* of pylethrombosis is not favorable, for even if, as in cirrhosis of the liver, venous collateral channels develop, for the purpose of conveying the portal blood to the general circulation, these will not suffice. The ascites increases progressively, and death appears unavoidable from asphyxia, cardiac paralysis, exhaustion, at times from excessive hemorrhage.

The *anatomic alterations* are readily recognizable. On dividing the trunk of the portal vein this vessel will be found occluded by a fibrinous thrombus which at times may be followed far into the intrahepatic or the peripheral branches. Mural thrombi that have occasioned only stenosis of the portal vein are less common. In addition the changes due to portal stasis are present.

Treatment.—The treatment of portal thrombosis is mechanical. **Abdominal puncture** should be performed when the ascites becomes excessive. From internal remedies, such as **digitalis, calomel, diuretin, urea,** and other **diuretics,** and from **laxatives, diaphoretics,** or **drastics,** no permanent success can be hoped. Also absorption of the thrombus by medicaments cannot be effected.

PURULENT INFLAMMATION OF THE PORTAL VEIN (SUPPURATIVE PYLEPHLEBITIS).

Etiology.—Suppurative pylephlebitis is a manifestation of **septicopyemia.** It develops in the sequence of inflammation in the radicular distribution of the portal vein if bacteria gain entrance into the portal vein from this point, excite inflammation of the wall of the vein, and in connection therewith give rise to the formation of a purulent thrombus contaminated by bacteria. Among the more common causes are dysentery, puerperal fever, carcinoma of the uterus, salpingitis, oöphoritis, carcinoma of the rectum, proctitis and periproctitis, peritonitis, perityphlitis, abscess of the spleen, gastric ulcer, carcinoma of the stomach, and intestinal ulceration. In the newborn pylephlebitis has been observed in the sequence of inflammation of the umbilicus.

Anatomic Alterations.—The wall of the portal vein appears friable and readily torn in the diseased situation. On opening the vein a smeary, purulent, at times offensive-smelling, brownish thrombotic mass is found at this point. Often portions of the thrombus have become detached, and have found their way into the liver, where they give rise to new foci of inflammation and suppuration. Collections of pus form also in other viscera, particularly in the lungs. The **spleen,** as in many infectious diseases, is enlarged and soft.

Symptoms, Diagnosis, and Prognosis.—The disease pursues the course of a **septicopyemia.** Repeated chills occur, and are followed by fever and sweating. The liver and the spleen are enlarged. Pressure over the liver induces pain. At times it has been possible to feel the portal vein through the abdominal walls as a thick cord. Often there is marked jaundice. Not rarely foci of purulent inflammation appear in various parts of the body, as, for instance, the lungs, the pleura, the pericardium, and the joints. Under the most favorable conditions the *diagnosis* can be made only with some degree of probability. The *prognosis* is unfavorable, as recovery appears impossible.

Treatment.—Efforts should be made to maintain the forces of the body as fully as possible, particularly by the use of **alcoholics** and **stimulants.** In addition, an **ice-bag** should be applied over the liver.

ANEURYSM OF THE HEPATIC ARTERY.

But a small number of cases of aneurysm of the hepatic artery have been recorded. The condition can be recognized with certainty only when a swelling with expansile pulsation is palpable to the right of the median line, and over which a cardiac-systolic vascular murmur is audible. At times symptoms are present like those of biliary colic: Colicky attacks of pain, vomiting, and jaundice. In one case under my observation death resulted from internal hemorrhage, the cause of which was found on post-mortem examination to be a ruptured aneurysm of the hepatic artery.

The **prognosis** is unfavorable, for every aneurysm has a tendency to undergo enlargement and rupture.

The **treatment** would be confined to the relief of particularly distressing symptoms. If the diagnosis is positive, persistent **rest in bed** and the application of an **ice-bag** would be indicated.

VII. DISEASES OF THE PANCREAS.

The diagnosis of disease of the pancreas is exceedingly difficult, for, in the first place, the organ is so concealed as to be accessible to physical examination only under especially favorable conditions, and, besides, no symptoms are known to be peculiarly distinctive of disease of the pancreas.

Acute and chronic inflammation of the pancreas is thus far only of anatomic interest.

[Acute pancreatitis may be hemorrhagic, suppurative, or gangrenous. It is attended with pain in the superior abdominal region, usually of sudden onset and paroxysmal exacerbation; generally with constipation, although diarrhea may supervene; with abdominal distention, icterus, an anxious and pinched expression, a small and rapid pulse, and variable temperature. Death usually occurs in from two to five days, from collapse. In treatment morphin may be required for the relief of pain, and stimulants, possibly by enema, to overcome collapse. If the pancreas can be felt to be enlarged and the diagnosis is certain, operation and evacuation of septic material may be undertaken as soon as collapse has passed off.

Chronic pancreatitis probably arises by extension from adjacent disease. It may set in gradually and without pain, or with severe epigastric pain, associated with nausea and vomiting, or followed by jaundice and perhaps also by a feeling of chilliness or a rigor. Tenderness is usually present in the umbilical region, and sometimes swelling of the pancreas can be made out. Appetite is usually wanting; the taking of food is followed by epigastric discomfort, and loss of flesh and strength is marked. Albuminuria is common, and glycosuria may be present. The temperature is variable. At a late stage hemorrhages may take place into the skin and from the mucous membranes, with death from asthenia. In diagnosis gall-stones in the choledoch duct, carcinoma of the head of the pancreas or of the liver and the bile-ducts, and chronic catarrh of the bile-ducts must be taken into consideration. The only treatment is surgical.—A. A. E.]

In obese persons **fat-necrosis** occurs occasionally **in the pancreas,** and in connection therewith there may be **hemorrhage into the pancreas.** Under such circumstances death takes place amid signs of ***shock*** and **progressive** collapse, or the pancreas undergoes marked **increase in size, exerts** pressure upon the intestine, and gives rise to *ileus*, or rupture into the abdominal cavity takes place, with the development of usually fatal peritonitis.

At times **calculi** form in the excretory ducts of the pancreas—*pancreatic sialolithiasis*—and these may give rise to severe and painful attacks of pancreatic colic.

Pancreatic cysts have been operated upon recently in a number of instances.

Carcinoma of the pancreas may involve the tail, the body, the head, or the entire organ. Most commonly the head is the seat of the disease. Generally the new-growth is primary. The most reliable sign is the demonstration of a nodular tumor, somewhat above the level of the umbilicus, not connected with the stomach, the omentum, the colon, the kidneys, the liver, or the abdominal lymphatic glands. Carcinoma at the head of the pancreas often surrounds the portal vein and the choldeoch duct, and occasions irremediable ascites and jaundice. The prognosis is unfavorable and the treatment purely symptomatic.

VIII. DISEASES OF THE PERITONEUM.

INFLAMMATION OF THE PERITONEUM (PERITONITIS).

Etiology.—Inflammation of the peritoneum in human beings occurs almost unexceptionally as a result of the activity of **bacteria**, among which the Bacterium coli commune, the Streptococcus pyogenes, the Staphylococcus pyogenes albus and aureus, the pneumoniacoccus, the gonococcus, the Bacillus typhosus, the Bacillus tuberculosis, and some others have been demonstrated in the inflammatory lesions. Naturally it would be possible to excite peritonitis by means of strictly chemic poisons, as for instance, injections of oil of turpentine, tincture of iodin, and the like, into the abdominal cavity, but such an occurrence is only exceptionally to be anticipated in human beings.

Infection of the peritoneum is usually brought about through certain **contributory causes**, which formerly, when the significance of bacteria was not understood, were considered the active and causative factors of the disease. Among these the greatest importance was attached to *cold*. *Refrigeratory* (*rheumatic*) *peritonitis* is, however, among the less common varieties of the disease. It has been observed after sleeping upon damp ground, and particularly in women, at the menstrual period, following cold bathing. At times traumatism is followed by infection of the peritoneum and the development of ***traumatic peritonitis***.

Falls, blows, shocks, crushing, and punctured wounds of the abdomen, may be mentioned as particularly common causes of traumatic peritonitis.

Peritonitis by extension probably occurs most commonly, and particularly in the sequence of inflammatory and ulcerative processes in any of the intestinal viscera, and in the vicinity of the peritoneum. It will suffice to mention a number of the more common disorders: ulcer, abscess, and carcinoma of the stomach, ulceration of the intestine, dysentery, carcinoma of the intestine, intestinal invagination and strangulation, typhlitis, appendicitis, abscess, carcinoma and echinococcus of the liver, abscess, carcinoma, echinococcus and infarction of the spleen, abscess and embolism of the kidney, paranephritis, impacted renal or biliary calculi, metritis, salpingitis, oöphoritis, cystitis, prostatitis, strangulated hernia, suppurating inguinal glands, burrowing abscesses of the vertebræ, pleuritis, pericarditis, etc. These and similar diseases may cause peritonitis by rupture and evacuation into the abdominal cavity of material capable of exciting inflammation. Such a condition is designated *perforative peritonitis*.

At times peritonitis develops in the course of **infectious diseases**, as, for instance, septicopyemia, erysipelas, fibrinous pneumonia, typhus fever, and the like. Without doubt **morbid alterations in the bodily metabolism** diminish the resistance of the peritoneum, and increase its susceptibility to inflammation. Among such disorders are uremia, pulmonary tuberculosis, the carcinomatous cachexia, and scorbutus. Peritonitis may be superadded to other diseases of the peritoneum, particularly *tuberculosis* and *carcinoma*. There are, however, cases in which no cause for the inflammation can be demonstrated—*cryptogenetic peritonitis*. The condition under such circumstances may often be an *alimentary peritonitis*, by which it is understood that bacteria migrate from the intestinal contents through the intestinal wall into the peritoneal cavity and give rise to inflammation. Not at all rarely the statement is positively made that the symptoms of peritonitis have rapidly developed in the sequence of **errors in diet** (the ingestion of cold or spoiled beer, or of unripe fruit).

Peritonitis may occur at any period of life; it may develop even in the newborn in the sequence of septic infection of the umbilical wound. At times children are born with chronic syphilitic peritonitis. Peritonitis is most common, naturally, between the *fifteenth and the forty-fifth year of life*. *Women* are attacked more commonly than men, because diseases of the female generative apparatus often constitute the source for inflammation of the peritoneum.

Anatomic Alterations.—Inflammatory processes in the peritoneum pursue much the same course as inflammatory processes in other serous membranes, and, accordingly, a differentiation has been made between **fibrinous, serous, purulent,** and **hemorrhagic peritonitis.** In addition there occurs **putrid peritonitis,** which results from a purulent peritonitis when the purulent exudate becomes infected with the bacteria of putrefaction. Often **mixed varieties** of peritonitis occur, which may be designated serofibrinous, fibrinopurulent, etc., respectively. In accordance with the extent of the inflammation of the peritoneum a distinction is made between *circumscribed* (*localized*) and *diffuse peritonitis.* Further reference to circumscribed peritonitis will not be made here, because this is considered in other places as perityphlitis, perinephritis, perihepatitis, perisplenitis, etc.

Fibrinous peritonitis is characterized by vivid redness and over-distention of the diseased peritoneum with blood. Here and there individual blood-vessels have ruptured, and punctate and sometimes larger extravasations of blood have taken place. Soon swelling, fatty degeneration, and desquamation of the endothelial cells of the peritoneum take place, and the serous membrane in these situation appears turbid and dull, like a plate of glass that has been breathed upon. The alterations in the endothelium render it possible for the exudate from the blood-vessels —containing considerable fibrin—to coagulate upon the surface of the peritoneum, and to form at first thin, cob-web like, and subsequently thicker, yellow, and opaque deposits. Through these fibrinous membranes numerous adhesions take place between the abdominal viscera, particularly loops of intestine. Should the process terminate in recovery, the fibrinous exudate may be entirely absorbed and disappear. More commonly, it is true, the exudate is more or less extensively transformed into connective tissue, which forms peritoneal bands and adhesions between the individual abdominal viscera. At times the entire mass of intestine and the remaining abdominal viscera are so firmly bound together that the whole constitutes an inseparable convolution, and the abdominal cavity is completely obliterated—*obliterative peritonitis.*

Serous peritonitis is attended with an accumulation of slightly turbid, yellowish or yellowish-green fluid in the abdominal cavity. As a rule, the fluid contains flocculi of fibrin, and also the intestines and the remaining abdominal viscera are covered with fibrinous masses, so that the condition generally becomes one of serofibrinous peritonitis. *Purulent inflammation of the peritoneum—suppurative peritonitis*—occurs far more commonly than serous peritonitis, and this fact is probably indicative of the greater irritability of the peritoneum in comparison with some other serous membranes (pleura, pericardium). The peritoneum is rather comparable with the meninges in this connection. The pus in the abdominal cavity presents the usual appearance, and it often reaches [illegible] in amount. It becomes especially abundant in the true pelvis. In addition to pus fibrinous deposits are also encountered, and these often bind the loops of intestine together. If adhesions are separated, deep pockets are often exposed filled with pus. Should absorption of the pus take place in the further course of the curative process, fibrinous deposits remain and subsequently undergo the same changes that have been described for fibrinous peritonitis. *Putrid peritonitis* is characterized by the offensive odor of the pus, being irritating to both the nostrils and the eyes. The putrid material causes irritation of the skin of the dissector. On microscopic examination of the pus the pus-corpuscles are found in a state of partial degeneration. The pus contains innumerable bacteria. Putrid peritonitis occurs, in addition to septicopyemia, particularly in the sequence of rupture of the abdominal viscera. If the ruptured organ is a gas-containing viscus (esophagus, stomach, intestine), gas frequently escapes into the abdominal cavity, and the condition is designated *pneumoperitonitis*. *Hemorrhagic peritonitis* occurs in connection with tuberculosis and carcinoma of the peritoneum, nephritis, and scorbutus.

Symptoms and Diagnosis.—The symptoms of peritonitis vary in accordance with the nature of the inflammation. An account of the symptoms of diffuse, acute, purulent peritonitis, perforative peritonitis, and chronic peritonitis follows:

Symptoms of Acute, Diffuse, Purulent Peritonitis.—A constant symptom of acute, diffuse, purulent peritonitis is *pain*. At first this may be purely local in the situation of the organ from which the inflammation of the peritoneum has originated, but soon it extends over the entire abdomen, and increases in such degree that the gentlest manipulation of the abdominal walls and the slight pressure of a thin sheet cannot be borne. The patient avoids speaking in a loud tone, deep breathing, or change in posture, in order to prevent aggravation of the pain. Not rarely, in spite of all precautions, the pain exhibits paroxysmal and colicky increase in severity, and this is frequently associated with audible gurgling in the abdomen and increased intestinal movement.

The *abdomen* is distended like a drum and at times to the point of rupture, the overlying skin being glistening and smooth. The cause for this condition consists in marked distention of the intestine with gas—intestinal meteorism—and this in turn is dependent upon disturbed intestinal digestion and abnormal fermentation of the intestinal contents. On *percussion of the abdomen* a uniformly loud tympanitic percussion-note is not rarely audible everywhere. In other instances areas of dulness appear in irregular distribution, in correspondence with accumulations of fluid exudate. Such dulness is most common in the dependent and lateral portions of the abdomen. Displacement of dulness with change in posture is scarcely observed, as the exudate is usually confined within circumscribed cavities. As a rule, fluctuation cannot be elicited at all over exudates, or but imperfectly. In accordance with the degree of meteorism the diaphragm, the lower borders of the lungs, and the heart are greatly displaced upward. The patient, therefore, complains frequently of *dyspnea*, a sense of *fear*, of *oppression*, and of *palpitation of the heart*.

Vomiting usually occurs in consequence of irritation of the stomach. The vomited matter consists at first of the gastric contents, and its appearance accordingly varies with accidental conditions; but later it frequently presents a greenish, bilious appearance. With progressive exhaustion the vomiting ceases, and it is often replaced by troublesome *eructation* or *singultus*. *Thirst* is greatly increased, whereas the *appetite* is completely wanting. The *tongue* is often coated, but in other instances it presents a clean and strikingly red appearance, usually in consequence of frequent vomiting and slight irritation of its mucous membrane. At times the *breath is offensive*. At the beginning of the disease the *stools* are generally thin and liquid, but subsequently marked constipation commonly exists. The urine is usually voided in diminished amount. The secretion is generally dark red (high-colored, saturated), frequently precipitates a brick-dust sediment, and contains much indican in consequence of derangement of intestinal digestion and increased decomposition of proteids in the intestine. Slight albuminuria also is not a rare occurrence. At times the patients complain of pain in the evacuation of urine and of retention of urine, symptoms that are often associated with pericystitis.

The *bodily temperature* is elevated at times as high as 40° C. (104° F.) and above, although the course of the fever is irregular. *Pulse* and *respiration* are frequently more greatly accelerated than the height of the fever warrants, because, in addition to the elevation of temperature, displacement of the lungs and of the heart and nervous influences are operative. The rapidity and the severity with which the *general condition* suffers are noteworthy. The facial expression is painful and the features are distorted, the eyes become deeply sunken and surrounded by blue lines, and the chin and nose become sharply prominent.

The *course of acute diffuse peritonitis* may terminate fatally within a few days amid septic manifestations and progressive asthenia, while in other instances the disease is prolonged for weeks and even for months, with numerous fluctuations, pursuing rather a subacute and even a chronic course. Often the outlook for recovery appears most promising, but a renewed turn for the worse rapidly dispels hope. Among *complications* symptoms of *ileus* may be mentioned first, and amid which death may take place without mechanical obstruction of the bowel being found upon post-mortem examination. The condition is therefore designated *dynamic ileus*, resulting from local paralysis of the musculature of the bowel.

At times metastatic inflammatory processes appear in other situations; most commonly in the right pleural cavity, and at times also in the pericardial cavity. Under such circumstances it is often remarkable within how short a time a pleuritic effusion may be again completely absorbed when the peritonitis shows a disposition toward improvement. Particularly serious are *circumscribed collections of pus*, which form especially between the loops of bowel. In the first place, they may readily give rise to renewed exacerbations of the peritoneal inflammation; and besides, they may rupture into adjacent organs, as, for instance, into the intestine, the stomach, the genito-urinary passages, through the abdominal walls, and particularly through the umbilicus. Although natural recovery may follow such an accident, it is also possible for the pus to become infected with fecal matter or with urine and become putrid, and in this way cause death by septicopyemia. Rupture of pus into the large blood-vessels of the abdominal cavity is dangerous, for in addition to hemorrhage general septicemia may occur. Also when, contrary to experience, absorption of pus takes place, serious *sequelæ* at times develop. The bowel may be so greatly displaced and distorted in consequence of peritoneal adhesions that *stenosis* and even *obstruction—ileus*—result. Similar conditions may also be brought about by loops of intestine slipping beneath peritonitic bands or being surrounded by them.

Symptoms of Perforative Peritonitis.—The symptoms of perforative peritonitis depend upon whether the inflammation of the peritoneum is free or encapsulated, and whether the rupture has taken place from a gas-containing or an airless viscus. In case of rupture of the stomach, the intestine, or the esophagus, in addition to particles of food, gas usually escapes into the abdominal cavity, and accordingly there develops a free or a sacculated pneumoperitonitis. With the development of a *free pneumoperitonitis* the patient not rarely cries aloud that something has torn within the abdominal cavity, and he complains generally of indescribable *pain in the abdomen*. The *abdominal walls* are retracted, stretched with board-like hardness, and exceedingly tender to touch. It is noteworthy that outlines of *loops of intestine* beneath the abdominal walls are not visible, and that also no peristaltic movement of the bowel can be detected. Of particularly great diagnostic significance is the disappearance of the *hepatic* and the *splenic dulness*, which results in consequence of the rise of the escaping air and the displacement of the liver and the spleen from the thoracic and abdominal walls. This important sign will be absent only when the liver and the spleen have previously been immovably bound by peritoneal adhesions to the thoracic and abdominal walls. Frequently *vomiting* occurs, although its absence has been observed in case of rupture of the stomach, because at times in the efforts at vomiting the contents of the stomach pass more readily through the site of rupture into the abdominal cavity. The general condition is characterized in an unmistakable manner by the signs of most profound *exhaustion*. The skin feels cool, the pulse is small and running, the voice is faint and hoarse, the face is pale, the eyes are sunken, retracted, and surrounded by gray rings, and the nose, the malar bones, and the chin stand out prominently. *Consciousness* is preserved. *Death* may result from putrid infection and progressive exhaustion within a few hours. At times it occurs as the result of asphyxia if the diaphragm, the lungs, and the heart are displaced upward excessively in consequence of intestinal and peritoneal meteorism. *Recovery* as a result of internal medication and expectancy occurs most rarely, but life has been saved in a number of instances of late by surgical intervention.

Sacculated pneumoperitonitis occurs especially in connection with ulceration of the stomach and the intestine, when rupture of these viscera has been preceded by circumscribed peritonitis with adhesions, so that the escape of gastric or intestinal contents takes place into the previously closed cavity. The condition is exceedingly difficult of recognition. Circumscribed tenderness in the abdominal cavity should be looked for. Over this area a dull note will be elicited on percussion, in so far as the exudate is in contact with the abdominal walls, whereas a tympanitic percussion-note will be obtained over the gas-containing cavity. The fact is of especial importance if displacement of the percussion-boundaries in accordance with the position of the body can be demonstrated, under which conditions the tympanitic percussion-note will always correspond with the highest point of the gas-containing cavity. It may also be possible that vigorous agitation of the body will elicit a *succussion-sound* or a *splashing sound*, although there will be some hesitation in subjecting the patient to such active manipulation. The formation of a sacculated pneumoperitonitis may take place so insidiously, as, for instance, in a case of typhoid fever, that even when the condition of the patient is most carefully followed the presence of the disease may perhaps be recognized only at the autopsy. The subject of subphrenic pyopneumothorax has been considered on p. 167.

If an airless viscus has ruptured into the peritoneal cavity (abscess of the liver or of the spleen, softened lymphatic glands, paranephritis, burrowing abscesses, and the like), or if an air-containing viscus has ruptured, and gas has accidentally not escaped into the abdominal cavity, an *acute diffuse peritonitis* develops within the shortest possible time, if free rupture has taken place into the abdominal cavity. That the condition is one of perforative peritonitis will be indicated by the fact that the patients often designate with absolute positiveness a circumscribed area as the source of their trouble, and that not rarely also in the further course of the disease definite local disturbances become more pronounced in this situation, as, for instance, increased tenderness and greater accumulation of exudate.

Sacculated perforative peritonitis will be recognized from the presence beneath the abdominal walls of circumscribed dulness, which yields increased resistance and pain on palpation. As the condition may develop quite insidiously, it at times escapes diagnosis. Occasionally it is recognized only from the sequelæ, if rupture of the encapsulated pus takes place through the abdominal walls or into intestinal viscera.

Symptoms of Chronic Peritonitis.—Among the chronic varieties of peritonitis peculiar clinical interest attaches to that which is attended with the formation of a serous, freely movable accumulation of fluid in the abdominal cavity—*chronic-serous peritonitis*. Usually without obvious cause more and more fluid gradually accumulates in the abdominal cavity. The *abdomen* increases progressively in size. *Fluctuation* can be elicited, and on percussion in the dorsal decubitus a tympanitic note is obtained over the upper portion of the abdomen corresponding to the loops of intestine floating upon the fluid, with dulness in the lateral portions. *Change in position* is followed by displacement of the percutory phenomena, the uppermost portion of the abdomen yielding a tympanitic percussion-note, while a dull note is obtained over the lowermost portion. The intestines always tend to rise to the surface of the fluid. The physical phenomena are, on the whole, the same as those that attend ascites. *Differentiation from ascites* is possible only from the fact that causes for this condition are not demonstrable, and that the inflammatory fluid is characterized by the presence of a greater amount of albumin (more than 4 per cent.) and a higher specific gravity (above 1014). Also tenderness of the abdominal walls and transient febrile movement are indicative of serous peritonitis. The differentiation from *tuberculous-serous peritonitis* is far more difficult. In favor of the latter disease would be the presence of tuberculosis in other organs (particularly the intestine, the lungs, the lymphatic glands, the kidneys, the epididymis, the urinary passages), or the detection of the thickened tuberculous omentum as a dense band passing transversely across the abdominal cavity. Even after the abdomen has been opened the diagnosis may still be doubtful, because at times nodular fibrous thickenings form upon the peritoneum in cases of chronic-serous peritonitis, presenting exactly the appearance of tubercles, and only differing from these histologically and bacteriologically. Chronic-serous peritonitis may pursue an afebrile course, and not rarely causes no other pain than that due to tension. Should the effusion of fluid be considerable, dyspnea and acceleration of cardiac action occur in consequence of displacement of the diaphragm, the lungs, and the heart. The disease not rarely extends over many months, undergoes exacerbations and remissions, and is susceptible of cure.

Prognosis.—The prognosis of all varieties of peritonitis is serious. Acute and perforative peritonitis must be considered particularly dangerous diseases, frequently causing death from exhaustion and septicemia, although the outlook for recovery has recently been improved in some varieties by surgical treatment.

Treatment.—The treatment of peritonitis depends upon the *variety of inflammation.* In cases of *acute, diffuse, purulent peritonitis* the patient should be advised to observe as complete rest as possible, and invariably to use a **bed-pan** in the evacuation of the bladder and the bowels. The **diet** should be liquid and consist preferably of milk, coffee with milk, tea with milk, beef-soup, and wine diluted half with water. For the relief of the severe thirst, and in the presence of violent vomiting, small **bits of ice** may be sucked. For the inflammatory alterations in the abdominal cavity, not too heavy **ice-bags** should be applied to the abdomen. When the acute inflammatory symptoms moderate the ice-bag should be replaced by **hot cataplasms**, which are more agreeable to most patients, and favor absorption of the effusion. Severe localized pain in the abdomen not rarely subsides speedily after the application of from 5 to 10 **leeches** to the abdomen. Among internal remedies the use of **opium or morphin** may be recommended:

℞ Powdered opium,	0.03 (½ grain),
Sugar,	0.3 (4½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

℞ Morphin hydrochlorate,	0.3 (4½ grains);
Glycerin,	
Distilled water,	each, 5.0 (75 minims).—M.

Dose: 0.5 (8 minims) once or twice daily injected subcutaneously.

Both morphin and opium are employed to place the bowel at rest. As a result the pain becomes less, and, besides, dissemination of the pus throughout the entire abdominal cavity is prevented. Some patients do not bear opium well, and develop severe pain, nausea, and delirium after its use, and for these morphin is better adapted. Opium or morphin should be given until the abdomen becomes entirely free from pain on pressure. Many weeks may be required to attain this end. Should improvement in the condition take place, the greatest care must be taken with regard to the diet and to getting up.

The treatment of *perforative peritonitis* is the same as that just described in so far as internal measures are applicable. On account of the alarming loss of strength it will be necessary to make frequent use of **stimulants**, as, for instance:

℞ Camphorated oil, 10.0 (2½ fluidrams).
Dose: 1.0 (15 minims) subcutaneously from three to six times daily.

If the perforation has involved the esophagus or the stomach, the introduction of all food or drink through the mouth should be interdicted, and the administration of nourishment be confined to rectal enemata of milk, meat-soup, and eggs (p. 197). Although internal treatment of perforative peritonitis affords only slight hope of recovery, it has been possible of late to bring this about in numerous cases through **surgical treatment.** The earlier operation is undertaken and the fewer the symptoms of general septicemia present the more successful will be the result. The operation generally consists in abdominal section—celiotomy—freeing the abdominal cavity and the abdominal viscera from the matters that have escaped through the perforation by careful sponging, and closure of the line of rupture by suture. At times it is necessary in the presence of ulcerative processes in the stomach or the bowel to precede suture by excision of the ulcer, or even by resection of portions of either viscus.

In a case of *chronic-serous peritonitis* a **milk-diet** should be prescribed (from 1½ to 2 quarts of boiled milk, to be swallowed in small amounts at short intervals), as this not rarely stimulates active diuresis and thereby favors absorption of the exudate. Locally I have observed good results in a number of instances from **inunction of the abdominal walls with green soap.** Among diuretics, **calomel, diuretin,** and preparations of **caffein** are particularly worthy of trial, while I have rarely observed good results from the use of urea:

℞ Mercurous chlorid,
Sugar, each, 0.3 (4½ grains).—M.
Make 10 such powders.
Dose: 1 powder thrice daily.

℞ Diuretin, 1.0 (15 grains);
Sugar, 0.5 (7½ ").—M.
Make 10 such powders.
Dose: 1 powder every three hours.

℞ Caffein sodio-salicylate, 1.0 (15 grains);
Sugar, 0.5 (7½ ").—M.
Make 10 such powders.
Dose: 1 powder thrice daily.

When calomel is used the mouth should be rinsed with **potassium chlorate** (10.0 : 200—2½ drams : 6½ fluidounces) after each meal in order to prevent the development of mercurial stomatitis. **Diaphoretics** are also worthy of trial; as, for instance, hot-air baths by means of a *sweating chamber,* and subcutaneous injections of ***pilocarpin hydrochlorate.***

℞ Solution of pilocarpin hydrochlorate, 0.2 : 10.0
(3 grains : 2½ fluidrams).
Dose: 0.5 (8 minims) subcutaneously.

The use of **purgatives** and **drastics** should be **abstained from.** Should there be danger of suffocation from excessive accumulation of fluid, **puncture** or **incision** of the abdomen should be practised, but not rarely fluid reaccumulates.

ABDOMINAL DROPSY (ASCITES).

Etiology.—Ascites consists in a collection of serous fluid in the peritoneal cavity, not resulting from inflammatory processes, but dependent upon purely physical causes. The fluid is not an exudate, but a *transudate.* Two modes of origin for such a transudate are recognized: namely, **increased blood-pressure in the veins** and **abnormal permeability of the walls of the veins.** Accordingly, a distinction is made between *hypostatic ascites* under the first conditions and *cachectic ascites* under the second. Hypostatic ascites either is a result of general venous stasis in connection with chronic disease of the heart or the respiratory organs, or it is due rather to local causes and develops in consequence of *portal stasis.* The last-named condition arises most commonly in connection with chronic interstitial hepatitis, and less commonly in connection with other diseases of the liver (carcinoma, abscess, echinococcus), with pylethrombosis and obstruction of the portal vein by adjacent tumors, lymphatic glands, and the like. Rarely **stenosis of the hepatic veins** constitutes a cause for hypostatic ascites. *Cachectic ascites* occurs most commonly in association with nephritis, although it attends also carcinoma, pulmonary tuberculosis, chronic suppuration, chronic diarrhea, and all debilitating conditions. Ascites occurs at all periods of life. It may develop during fetal life in consequence of hereditary syphilis, and cause obstruction in labor. Most commonly it is observed after the twentieth year of life, as a large number of its causes first become operative at about this time.

Anatomic Alterations.—Extensive ascites gives rise to **distention of the abdomen,** while smaller accumulations of fluid, on the other hand, collect first in the true pelvis. Under the conditions first named the fluid often escapes in a powerful stream when the abdomen is opened, as the amount of fluid may exceed twenty quarts. As a rule, the fluid is greenish yellow in *appearance,* seldom bloody, but in the presence of jaundice it contains biliary coloring-matter. The fluid is transparent and clear, and contains at most a few grayish flocculi of detached endothelial cells involved in fatty degeneration, and soft gelatinous coagula, which may after a time undergo solution again. At times glistening crystals of cholesterin are present. The *reaction* of the fluid is alkaline, and its *specific gravity* fluctuates between 1004 and 1014. When ascites has existed for some time the abdominal organs with which the fluid comes in contact often appear pale and macerated.

Fatty ascites and *chylous ascites* have further been described. Fatty ascites were better designated *fatty peritonitis*. It develops at times in cases of tuberculosis or carcinoma of the peritoneum, when a considerable number of fattily degenerated cells are thrown off by the tuberculous nodules or the carcinomatous new-growth, and become in part admixed with the peritoneal exudate and in part dissolved therein. The fluid presents a milky, opaque appearance, and a layer of fat forms upon its surface upon standing. *Chylous ascites* likewise is attended with fluid of milky appearance, but this results from admixture with chyle, which is derived from ruptured lacteals. In contradistinction from fatty ascites, the chylous fluid contains sugar. The condition is most common in association with carcinoma of abdominal viscera.

Symptoms and Diagnosis.—The principal symptom of ascites consists in the presence of a free, movable accumulation of fluid in the abdominal cavity, which, in contradistinction from peritonitic fluid, has developed without fever and without noteworthy pain. Transudation of considerable amount in the abdominal cavity causes **increase in the size of the abdomen,** which at times attains enormous proportions. The anterior aspect of the abdomen appears flattened, while the lateral aspects are particularly expanded. In the erect posture the fluid accumulates in the lower half of the abdomen, and a sort of pendulous abdomen forms. The skin of the abdomen is smooth and glistening, and not rarely small points of rupture in the skin occur in the lower portions—so-called *striæ*—which entirely resemble the well-known cicatrices of pregnancy. Upon the posterior and lateral aspects of the abdomen cutaneous edema not rarely develops, and it may become so excessive as to cause rupture of the skin, with escape of fluid, erythema, erysipelas, or gangrene of the skin, and at times with death from septicemia. The **umbilicus** is either obliterated or it protrudes like a hernia. At the same time it is translucent in transmitted light, an indication that the protrusion contains fluid that has found its way out of the abdominal cavity through the patulous and palpable umbilical ring. The **cutaneous abdominal veins** are usually greatly distended and markedly tortuous, obviously because of compression and obstruction of the ascending vena cava by the abdominal fluid, so that a portion of the venous blood from the lower extremities must find its way to the heart through the veins of the abdominal wall. The demonstration of **fluctuation** is extremely important from a diagnostic point of view. The **percutory phenomena vary with the position of the body.** In the dorsal decubitus a tympanitic note will be developed over the upper anterior portions of the abdomen, and a dull note over the lower and lateral portions. In the lateral decubitus the dulness over the uppermost side disappears, and is replaced by a tympanitic percussion-note. The lowermost portion of the abdomen, on the other hand, yields a dull note everywhere. These phenomena depend upon the fact that the intestines, which yield a tympanitic note, constantly have a tendency to float upon the surface of the fluid.

In the dorsal decubitus a narrow, longitudinal band with a tympanitic percussion-note appears not rarely in the posterior and lateral portions, corresponding with the gas-containing ascending and descending colon. The tympanitic area over the anterior abdominal wall will be absent in association with ascites when the mesentery is short with relation to the amount of fluid in the abdominal cavity, so that the intestines are prevented from floating upon, but are immersed beneath, the surface of the fluid. Small accumulations of fluid in the abdominal cavity that have collected in the true pelvis become accessible to percussion only if the pelvis is elevated or the patient assumes the prone position. Under the condition first named, if the fluid enters the upper portion of the abdominal cavity, dulness appears upon either side of the vertebral column, and under the latter condition over the anterior abdominal wall.

Extensive ascites may occasion alarming dyspnea by displacement upward of the diaphragm, the lungs, and the heart, and all the more because the expansion of the lungs and the movements of the heart are often interfered with in consequence of associated hydrothorax and hydropericardium.

We have hitherto considered only ascites free in the abdominal cavity. At times, however, the effusion is *encapsulated* or *sacculated*, and the principal symptom then is circumscribed dulness and fluctuation. In contradistinction from circumscribed peritonitis, fever and pain are wanting.

In the presence of general causes for stasis and of cachectic ascites, cutaneous edema, hypostatic urine, and often also hydrothorax and hydropericardium are encountered. Should ascites exist alone and without edema in other situations, it is probably of portal origin. If the ascites by reason of its extent exerts considerable pressure upon the inferior vena cava, hypostatic edema in the lower extremities may eventually develop, but in comparison with large abdominal effusions this usually attains slight proportions. Ascites is generally a chronic disorder, as its etiologic factors are of a chronic nature. Frequently exacerbations and remissions alternate, accordingly as the strength of the muscular structure of the right side of the heart and in connection therewith the stasis in the distribution of the inferior vena cava vary. Death from suffocation and cardiac paralysis are the principal dangers.

On the whole, ascites is easily and certainly recognized. Although similar physical signs are yielded also by chronic-serous and tuberculous-serous peritonitis, stasis or cachexia will, in the first place, usually be found to be the cause of ascites; in addition, febrile movement and marked tenderness of the abdomen are wanting; and, finally, any fluid obtained upon puncture will be characterized by its lower specific gravity (below 1014) and the presence of a small amount of albumin (less than 4 per cent.). Even experienced clinicians have in numerous instances confounded ascites with an **ovarian cyst.** In the differential diagnosis it is important to recall that the anterior abdominal wall is flattened when ascites exists, while it is pushed forward by an ovarian cyst. The umbilicus is obliterated in the presence of ascites or it protrudes forward, while with ovarian cysts it remains unchanged, or is even displaced upward. With ascites the lateral aspects of the abdomen are dull on percussion when the patient occupies the dorsal decubitus, while the anterior portion yields a tympanitic note. The conditions are reversed when an ovarian cyst is present, with dulness anteriorly and a tympanitic note laterally. Variations in the percussion-phenomena in accordance with the position of the body are characteristic of ascites, and are wanting with ovarian cysts, as the fluid is not capable of free movement in the cyst, which is closed upon all sides. Although fluctuation is present in both diseases, it is confined strictly to the area of dulness with ovarian cysts, while with ascites it is appreciable also beyond this area. With ascites the uterus is often displaced downward, and is exceedingly mobile, in consequence of the pressure exerted by the collection of fluid, while with ovarian cysts it is displaced upward or laterally.

If fluid be obtained for examination by puncture, it will be found in the presence of ascites to present a lower specific gravity (below 1014) than in that of an ovarian cyst (above 1015), and that with the latter disease the fluid not rarely exhibits a gelatinous consistency, and that, finally, on microscopic examination the sediment will be found to contain squamous epithelial cells with ascites, while with ovarian cysts cylindric epithelial cells are present.

Prognosis.—The prognosis of ascites depends upon the possibility of controlling the causes of the disorder. As a rule, such control can be acquired temporarily in the most favorable cases, but ultimately the remedies employed fail, and the patient dies with excessive accumulation of fluid in the abdominal cavity as a result of asphyxia or cardiac paralysis.

Treatment.—The treatment of ascites should be, in the first place, **causal,** and in accordance with the causative factors *heart-tonics* (digitalis), *diuretics* (diuretin, calomel, urea), *drastics, diaphoretics,* or *stimulants* should be employed. Among measures of **local treatment** only one is trustworthy and to be recommended, namely, *abdominal puncture,* while faradization of the abdominal wall, inunctions with soft soap, abdominal bandages, and the like, are unreliable. As abdominal puncture is a harmless procedure, which is capable of affording the patient great relief, the custom is properly becoming more common of practising it early, and not reserving it as a last resort. As often as the fluid reaccumulates in troublesome or threatening degree puncture of the abdomen should be repeated. In rare cases, however, reaccumulation of fluid does not take place, and recovery ensues.

CARCINOMA OF THE PERITONEUM.

As a rule, carcinoma of the peritoneum is a *secondary disorder*, the new-growth extending directly from involved abdominal viscera to the adjacent peritoneum or discrete metastasis taking place. At times the peritoneum is strewn with innumerable small nodules suggestive of miliary tubercles—*miliary carcinoma of the peritoneum;* or extensive *carcinomatous nodules* develop; or, finally, the peritoneum is in places the seat of *carcinomatous infiltration.* The abdominal cavity generally contains inflammatory or edematous fluid, which may be serous, but is frequently bloody and at times fatty or chylous.

The **diagnosis** is generally based upon the discovery of **freely movable fluid** in the abdominal cavity (fluctuation, alterations in the percussion-phenomena with change in posture), and upon the conclusion that the condition is of carcinomatous origin from the discovery of **similar disease in other organs.** Should abdominal puncture be practised, the bloody appearance of the fluid obtained from the abdominal cavity, or its fatty or chylous appearance, and the occurrence of numerous fattily degenerated or polynuclear cells, will be noteworthy. In doubtful cases examination should be made for *carcinomatous induration and enlargement of peripheral lymphatic glands*, particularly in the groin and above the left clavicle.

The **prognosis** is unfavorable and the **treatment** purely symptomatic.

ECHINOCOCCUS OF THE PERITONEUM.

Echinococcus of the peritoneum occurs either in conjunction with echinococcus in other abdominal viscera, particularly the liver, or in the peritoneum alone. Palpation discloses the presence of a fluctuating tumor, over which at times also hydatid tremor can be felt. Occasionally innumerable echinococcus-vesicles are present in the abdominal cavity, and in other instances but a single echinococcus-cyst has developed, as, for instance, in the omentum. In the latter event the cyst could be removed by surgical means. The principal danger consists in progressive growth and asphyxia.

PART IV.

DISEASES OF THE GENITO-URINARY ORGANS.

I. DISEASES OF THE KIDNEYS.

DIAGNOSTIC PRELIMINARY CONSIDERATIONS.

MOST diseases of the kidneys can be recognized from the *alterations in the urine* to which they give rise. Only rarely are peculiarities discovered by local examination of the kidneys. Among the urinary changes the *character of the sediment* (which is best obtained by means of a centrifuge) and admixture of albumin or of blood—*albuminuria* or *hematuria*—are the most significant. The importance of the two phenomena last mentioned will justify their more thorough consideration. A description of *uremia* will be added, because this represents a symptom-complex that is observed with especial frequency in connection with diseases of the kidneys.

ALBUMINURIA.

The appearance of albumin in the urine—albuminuria—must always be considered a **morbid manifestation**, for, although it may be possible to obtain small amounts of albumin from large amounts of urine from healthy persons, the urine of a healthy individual appears always to be free from albumin on employment of the usual clinical methods of examination.

A condition of **physiologic albuminuria** has been described, in which albuminuria has appeared in apparently healthy persons after walking long distances, bodily over-exertion of other kinds, emotional disturbances, cold baths, the ingestion of eggs; but whether in these cases the kidneys were in reality perfectly healthy does not appear to have been demonstrated with certainty. In any event, too much caution cannot be observed in admitting the existence of physiologic albuminuria, for not rarely undoubted disease of the kidney may make its appearance some time later.

Pathologic albuminuria is by no means always associated with disease of the kidneys. The urine may be excreted in a healthy state from the kidneys and become albuminous subsequently from admixture with pus or blood in the urinary passages. A distinction can, therefore, be made between *renal* and *accidental albuminuria*, the latter being designated—and, in my opinion, inappropriately—by French clinicians also *false albuminuria*. *Renal albuminuria* depends, in the first place, upon functional disturbance in the epithelial cells of the blood-vessels in the Malpighian bodies, whose duty it is to prevent the passage of albumin from the blood into the urine. The epithelial cells of the convoluted uriniferous tubules also appear to exhibit similar activity. Even slight circulatory and nutritive disturbances are capable of inhibiting the activity of the structures named without the presence necessarily of profound anatomic alterations. Renal albuminuria may be the result of primary disease of the kidneys, or the kidneys may only be involved subsequently as a result of alterations in the blood, so that, accordingly, a *nephrogenous* and a *hematogenous albuminuria* can be considered. Often a sharp distinction is scarcely possible.

While albuminuria may persist in some diseases for months and years, in others it is but transitory. In the first event, the condition can be described as *persistent*, and in the latter as *transitory albuminuria*. At times albuminuria occurs daily, or at definite other intervals within certain hours of the day, and it is then designated *cyclic*. Not rarely only the urine of the day contains albumin, while that of the night is free from it, and this difference has been attributed to the influence of bodily movement.

For the *demonstration of albuminuria* the **boiling-nitric-acid test** and the **nitric-acid test** of Heller will suffice in most cases. Should further confirmation be desired, the **picric-acid test** of Galippe, the test with yellow **potassium ferrocyanid and acetic acid** of Boedecker, or the test with **acetic acid and sodium sulphate** of Panum may be employed. With all tests for albumin the urine should be perfectly clear, and the specimen should, therefore, be filtered. If the urine contains bacteria, it will generally not be clear even when filtered. It should then be shaken with heavy magnesium oxid, and then filtered.

In employing the *boiling-nitric-acid test* the urine is introduced into a test-tube and is boiled. If the urine is acid, and if it remains clear on boiling, it contains no albumin. If, however, a turbidity ensues, this may depend upon earthy phosphates, which have been precipitated on heating the urine, or upon coagulated albumin. If on addition of nitric acid in excess the turbidity disappears, it has been dependent upon earthy phosphates. If, however, it persists, or even increases, it is dependent upon albumin, and albuminuria exists. Dilute acetic acid (1 per cent.) may be employed instead of nitric acid, but great care must be observed in the addition of acetic acid, as an excess may cause the formation of acid albumin, which is

Heller's *nitric-acid test* is an exceedingly delicate and commendable test for the presence of albumin. Several cubic centimeters of pure nitric acid are introduced into a test-tube, and upon this the urine is carefully permitted to flow along the sides of the tube so as to form in a layer upon the surface of the acid. If albumin be present, a white, sharp, readily recognizable band will appear at the line of contact. At times a second ring of urates appears at a somewhat lower level, but this, in contradistinction from the ring of albumin, disappears on application of heat. Urine containing much indican forms at times a brown ring, which is readily and clearly distinguishable from the ring of albumin by its color and transparency.

In the performance of the *picric-acid test* of Galippe the solution of picric acid and citric acid—so-called Esbach's reagent—may be advantageously employed:

℞ Picric acid,	10.0 (2½ drams);
Citric acid,	20.0 (5 ");
Distilled water,	1000.0 (32 fluidounces).—M.

Esbach's reagent for albumin.

The presence of albumin in the urine will be indicated by the immediate appearance of a permanent turbidity on addition of Esbach's reagent in excess. If the reagent be added only drop by drop, each drop as it falls into the urine causes turbidity, but this disappears on agitation until picric acid has been added to the urine in excess. Care should, further, be taken that the urine does not contain quinin, antipyrin, thallin, and potassium-salts, as these likewise yield a precipitate with picric acid.

Boedecker's *test for albumin with yellow potassium ferrocyanid and acetic acid* is exceedingly delicate. To several cubic centimeters of urine in a test-tube about the same amount of a concentrated solution of yellow potassium ferrocyanid is added, and to the mixture concentrated acetic acid drop by drop. The presence of albumin is indicated by the turbidity that appears with the addition of each drop of acetic acid.

Panum's *test for albumin with a saturated solution of sodium sulphate and acetic acid* (instead of sodium sulphate, sodium chlorid or magnesium sulphate also may be employed) is equally delicate. Approximately equal parts of urine and the solution of sodium sulphate are mixed in a test-tube, and then several drops of concentrated acetic acid are added until the urine acquires a highly acid reaction. If the mixture be heated, turbidity will occur if the urine contains albumin.

A number of other tests for albumin have been recommended (metaphosphoric acid, trichloracetic acid, etc.), but these are not indispensable to the practitioner.

The tests for albumin in common clinical employ are applicable to the two varieties of albumin particularly concerned in diseases of the kidneys, namely, *serum-albumin* and *serum-globulin*. There is scarcely any need on the part of the general practitioner to differentiate these two forms of albumin—which, as is well known, form the principal albuminous bodies of the blood-plasma—and to examine them separately. At times other albuminous bodies appear in the urine, namely, *albumoses* (*propeptones*), and at times also *nucleo-albumins*. Until recently it was believed that *peptones* also could be eliminated with the urine, but peptonuria does not occur in human beings if the chemic definition of peptones proposed by Kühne is accepted.

Albumosuria can be recognized from the fact that the urine remains clear on boiling and addition of nitric acid, and becomes turbid only on cooling. If the turbid urine be again heated, it will again become clear. A practical method of examination for albumoses consists in performing Heller's test for albumin, and, if a white ring of albumin forms, heating the test-tube. If the ring is due to albumoses, it disappears on heating; while if due to serum-albumin and serum-globulin it becomes more intense.

There is but one certain method of determining the *amount of albumin in the urine*, namely, boiling the urine, completely separating the albumin by careful addition of dilute acetic acid, and then weighing it, after previous drying upon a previously weighed filter. Such a procedure requires far too much time for the general practitioner, apart from the fact that a drying chamber and chemic scales are necessary for its application. It is, therefore, a matter for congratulation that Esbach has described a simple apparatus in the form of his *albuminometer*, by means of which the amount of albumin can be approximately estimated. Further, the determinations of albumin by means of carefully graduated Esbach albuminometers often agree remarkably with the results of weighing.

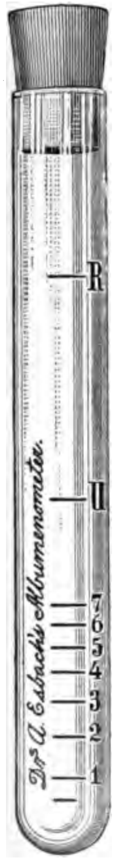

FIG. 55.—Esbach's albuminometer; natural size.

Esbach's *albuminometer* (Fig. 55) consists of a test-tube of thick glass, which can be closed by a rubber stopper. The tube bears two principal marks, the upper of which is indicated by the letter R and the lower by the letter U. Below the U-line is a scale down to the bottom of the tube, the lowermost figure of which is ½ and the uppermost 7. In using the instrument the test-tube is filled with clear, filtered urine to the line U (urine); then Esbach's reagent, consisting, as described, of picric acid and citric acid (p. 389) is added up to the line R (reagent), and with the opening closed the whole is inverted several times for the purpose of thorough admixture. The instrument is permitted to stand for twenty-four hours at room-temperature (14° R.—17.5° C.—63.5 F.). At the end of this time the level of the column of albumin that has been deposited at the bottom of the tube is read off. If, for instance, the level corresponds with the figure 3, this will indicate that 3 grams of albumin are present in 1000 c.c. of urine, therefore 0.3 per cent.

In using the albuminometer of Esbach the following facts should be borne in mind: A knowledge of the percentage of albumin will naturally be of value if in addition the daily amount of urine is known, and therefrom the total daily amount of albumin is estimated. The temperature of the room in which the albuminometer is kept should not be below 14° R. (17.5° C.—63.5°F.), as otherwise the albumin will not be precipitated. Now and then, although rarely, urine is encountered in which, in spite of the observance of all precautions, no precipitate of albumin takes place, but a cloud of albumin floats in the upper layers of the urine. Should the urine contain more than 0.7 per cent. of albumin, it should be diluted with an equal amount of water before it is introduced into the albuminometer. The patients shall not have used quinin, antipyrin, thallin, and potassium-salts, as picric acid, as has already been mentioned, forms precipitates with these substanees.

The amount of albumin eliminated with the urine is susceptible of great variation in accordance with the causes of the albuminuria. At times only traces of albumin will be present, with a scarcely appreciable turbidity of the previously clear urine, while in other instances the boiled urine may become a solid coagulum, which will not flow from the inverted tube.

On careful inquiry into the *clinical causes of albuminuria, hematogenous albuminuria* will be found, first, as a result of *circulatory disorders.* Both anemia and venous hyperemia of the kidneys are frequently attended with albuminuria. The *nervous albuminuria* that appears in the sequence of epileptic attacks, cerebral hemorrhage, tetanus, hysteria, progressive paralysis of the insane, etc., is often probably also due to disturbances in the renal circulation. Not rarely albuminuria is associated with *anemic* and *cachectic states,* although under such circumstances there is frequently a connection with hematogenous albuminuria, because in the course of the conditions named fatty degeneration readily develops, particularly in the epithelial cells of the convoluted uriniferous tubules. *Nephrogenous albuminuria* occurs particularly in association with *diffuse inflammatory conditions of the kidneys,* but also with all other possible diseases of the kidneys. *Febrile* or *infectious* and *toxic albuminuria* should further be mentioned as especial varieties of nephrogenous albuminuria. Febrile infectious diseases are frequently attended with albuminuria. The injury to the renal epithelium under such conditions is dependent probably less upon the elevation of bodily temperature than upon the infection, as afebrile infectious diseases may, likewise, give rise to albuminuria. Probably bacterial poisons (toxins) are the substances that cause irritation of the kidneys in the process of elimination with the urine. Toxic albuminuria may be induced by many substances, whether introduced through the mouth, the rectum, or the skin. Among these may be mentioned mineral acids, carbolic acid, mercuric chlorid, phosphorus, arsenic, lead, cantharides, balsamics, and irritating applications to the skin of all kinds. Albuminuria occurs also at times after burns of the skin and chronic cutaneous eruptions, in all probability induced by the absorption of toxic substances from the skin.

The danger from albuminuria does not reside in the loss of albumin, for an albuminuria with the loss of more than 20.0 grams of albumin daily is rare, and could easily be compensated for by the ingestion of several eggs or of meat. Therefore the **prognosis** *of albuminuria* depends solely upon the causative factors. When these are incurable so also is the albuminuria.

In the **treatment** *of albuminuria* the **nourishment** and the **mode of life** play the most important part. No medicine is known capable of curing albuminuria, and tannic acid, fuchsin, brombenzol, strontium lactate, and strontium bromid have been incorrectly recommended as such. Patients with albuminuria should avoid all *food* and *drink* capable of irritating the kidneys, particularly strong coffee and tea, alcoholics, sharp spices and acids, and asparagus. A milk-diet is particularly to be recommended: from 1 to 2 quarts of thoroughly boiled milk being taken in small amounts throughout the day. Meat should be taken only in moderate amount, and white meat (lamb, veal, poultry) particularly is to be preferred. Vegetables and light pastry may be recommended. Thirst should be relieved by acid or alkaline waters (Vichy, Bilin, Giesshübel, Ems, Fachingen, etc.). The patient should wear flannel next to the skin, and take twice weekly a bath at a temperature of 28° R. (35° C.—95° F.). All exposure to cold should be carefully avoided. In the winter the well-to-do should be advised to sojourn in an equable, warm **southern climate**. I have observed excellent results in a number of instances from a winter spent in Helouan, near Cairo. *Bodily over-exertion* should not be permitted under any circumstances, as it may cause a recurrence of a cured albuminuria.

HEMATURIA.

The presence of blood in the urine is designated hematuria when the urine contains sufficient hemoglobin to give rise to a distinctly bloody hue. Two varieties of hematuria must, however, be distinguished, accordingly as red blood-corpuscles or, after previous solution of red blood-corpuscles, only free hemoglobin is present in the urine. The latter condition is designated *hemoglobinuria*, as opposed to the more common occurrence of *hematuria*.

Hematuria is readily recognized from the distinctive color of the urine. In accordance with the number of red blood-corpuscles present in the urine, and the transformation of their contained hemoglobin into methemoglobin, the urine exhibits a light-rose color, suggestive of the appearance of meat-infusion, or presenting a blood-red, brownish, or even blackish appearance. Should any doubt exist as to the nature of the condition, the *urinary sediment* should be examined for red blood-corpuscles, or Heller's *blood-test* should be performed. The red blood-corpuscles in the urinary sediment, which often discloses the presence of blood by its brownish-color, are at times unchanged in form, although at other times (particularly in concentrated urine) they exhibit thorn-apple processes, or they appear swollen and biconvex, or lentil-shaped and even spherical. At times unusually small spherical red blood-corpuscles are encountered—so-called microcytes. Frequently red blood-corpuscles have yielded up their hemoglobin and appear as colorless discs of double contour—so-called blood-shadows. At times, particularly in the hot summer months, ameboid movement and constrictions can on careful observation be detected in isolated red blood-corpuscles. Should tube-casts appear in the urine in association with renal hematuria, they may be covered with red blood-corpuscles or be entirely constituted of them (blood-clasts). Tube-casts and epithelial cells from the uriniferous tubules also not rarely present a blood-stained or a brownish discoloration with hemoglobin. Rarely, hematoidin-crystals are present in urinary sediment. It is noteworthy that the red blood-corpuscles in the urine are almost never arranged upon one another in columns or in rouleaux, but are separated from one another, or lie in contact only at their borders.

Heller's blood-test is performed as follows: Several cubic centimeters of urine are introduced into a test-tube, and potassium hydroxid is added in excess until the urine acquires an alkaline reaction. If the mixture be heated, the earthy phosphates are precipitated on boiling in the form of coarse flocculi, which after a time are deposited upon the bottom of the tube. In urine free from blood this precipitate appears gray, while in urine containing blood it acquires a reddish, brownish, or brownish-black appearance. The test is so delicate that *spectroscopic examination of the urine* and *Teichmann's blood-test* are unnecessary.

Bloody urine is necessarily albuminous, as the hemoglobin consists of blood coloring-matter and proteid. So long as the condition is one of pure hematuria the urine contains only so much albumin as corresponds with the amount of blood present. The amount of albumin is increased when, in addition to hematuria, albuminuria also exists. The reaction of the urine depends upon the nature of the primary disease. In any event the presence of even quite a considerable amount of blood in the urine is never capable of rendering alkaline the naturally acid reaction of the urine.

The *causes of hematuria* reside in alterations either in the kidneys or in the urinary passages, and the character of the hematuria varies accordingly. With renal hematuria the blood is intimately admixed with urine, and retains the same color and character at the beginning and at the end of the act of micturition. Hemorrhages from the pelvis of the kidney and the ureters disclose their origin frequently from the appearance in the urine of cylindric blood-clots, which represent casts of the ureter. With hemorrhages from the bladder the urine first voided is frequently less bloody than that evacuated last, because the blood, by reason of its weight, has collected in particular abundance in the fundus of the bladder. Hemorrhages from the urethra, finally, are very slight, and often at the beginning of the act of micturition a few drops of blood make their appearance. With hemorrhages from the posterior portion of the urethra a few drops of blood are expelled only toward the close of the act of micturition, and often with painful tenesmus.

Renal hemorrhage occurs in connection with numerous *diseases of the kidneys*, as, for instance, acute nephritis, embolism of the renal artery, carcinoma of the kidney, renal tuberculosis, echinococcus of the kidney, and injury of the kidney. At times renal hematuria is dependent upon *alterations in the blood*, among the causes of which leukemia, pseudoleukemia, pernicious anemia, scorbutus, purpura, and hemophilia may be mentioned. At times hematuria occurs in the course of *infectious diseases* if these assume the so-called hemorrhagic character, and give rise to hemorrhages of all kinds. *Toxic hemorrhage from the kidney*, such as may be observed following the ingestion of cantharides, quinin, salicylic acid, potassium-salts, mineral acids, carbolic acid, etc., is worthy of especial mention. *Hemorrhages from the pelvis of the kidney and the ureter* are induced most commonly by renal calculi, less commonly by parasites, carcinoma, tuberculosis, or by rupture of an aneurysm of the renal artery.

Hemorrhages from the bladder occur in connection with cystitis, vesical calculi, carcinoma, tuberculosis, parasites, and in the sequence of injuries (external and internal, with the catheter). At times the veins surrounding the neck of the bladder undergo dilatation, forming so-called vesical hemorrhoids, which by rupture may give rise to profuse hemorrhage from the bladder.

Hemorrhage from the urethra occurs in connection with severe gonorrhea (so-called Russian gonorrhea), with impaction of urinary calculi and injuries with the catheter.

Hematuria is but rarely attended with such profuse loss of blood that life is endangered by hemorrhage. Should life be threatened, the danger will depend rather upon the causes of the hematuria. The latter influence is responsible also for the fact that hematuria is of variable duration, and either terminates with a single attack or is frequently repeated.

The **treatment** *of hematuria* must be directed to the causative conditions. Under all circumstances **bodily rest is indicated.** Not much can be hoped for from *hemostatics.*

Hemoglobinuria indicates the *occurrence of dissolved blood coloring-matter in the urine.* Hemoglobinuria is invariably preceded by hemoglobinemia; that is, red blood-corpuscles have first been dissolved in the blood and have given up their coloring-matter to the blood-plasma. Naturally the hemoglobinemia must have attained a certain degree of intensity before hemoglobinuria occurs, for the spleen, the liver, and the lymphatic glands are capable of taking up the dissolved hemoglobin from the blood-plasma and utilizing it if the hemoglobinemia be but slight. Hemoglobinemia and hemoglobinuria are most commonly caused by the action of toxic substances upon the blood, either of *chemic* or of *bacterial origin* (*toxins*). Among *chemic poisons* potassium chlorate particularly is well known as a solvent for blood-corpuscles and as a cause for hemoglobinuria. A similar influence is exerted by quinin, mineral acids, carbolic acid, nitrobenzol, aniline-oil, copper sulphate, hydrogen arsenid, etc. Hemoglobinuria can be induced in human beings by transfusion of blood from animals. Occasionally hemoglobinuria has been observed after the ingestion of *fungi*, particularly mussels. Among *infectious diseases*, in the sequence of which hemoglobinuria has been observed, typhoid fever, pharyngeal diphtheria, malaria, acute miliary tuberculosis, small-pox, septicemia, and syphilis may be named. The hemoglobinuria attending afebrile cyanosis of the newborn, and following burns of the skin, should further be mentioned. At times hemoglobinuria occurs in association with *diseases of the blood*, as, for instance, purpura and scorbutus. It has been observed also in the sequence of *sunstroke* and *fat-embolism*. *Paroxysmal hemoglobinuria* will be considered among the diseases of the blood.

In cases of hemoglobinuria the *urine* presents a *bloody appearance*, at times like that of fresh blood, but often reddish-brown, as the urine frequently contains an abundance of methemoglobin. The urine in a thin layer is transparent; it yields *Heller's test for blood*, although in the often brownish-discolored urinary sediment either no red blood-corpuscles at all are found or but a small number of usually discolored shadows, so that there will be no doubt that the condition is one of hemoglobinuria. Often the blood coloring-matter forms flocculent and filamentous brownish and greenish irregularly shaped masses, and tube-casts of compressed blood coloring-matter also appear. Such epithelial cells from the uriniferous tubules as may be present are frequently stained brownish or greenish by hemoglobin. At times crystals of hematoidin also are present. On boiling the urine a finely flocculent precipitate of albumin does not occur, but there results a large, coherent coagulum of albumin, stained brownish by hemoglobin. *Spectroscopic examination of the urine* reveals, in addition to the two bands of oxyhemoglobin between the Fraunhofer lines D and E, the bands of methemoglobin, of which especially that between the lines C and D is usually distinct. Only rarely are the bands of methemoglobin alone visible.

The **prognosis** of hemoglobinuria depends upon the causative condition, and the same statement is applicable also to the **treatment.**

Hematinuria has rarely been observed, as, for instance, after pharyngeal diphtheria. Within recent years attention has been directed to ***hematoporphyrinuria***, which has been observed particularly in connection with the toxic effects of sulphonal and trional, and with typhoid fever and mental disease. The prognosis is unfavorable.

UREMIA.

Etiology.—Uremia is a symptom-complex that is not rarely observed in the sequence of diseases of the kidneys. In order to avoid repetition, it will not be without utility to precede a consideration of the individual diseases of the kidneys by a description of uremia. The conditions for the development of uremia are always provided when the elimination of certain toxic substances with the urine is interfered with. This occurs most commonly when the amount of urine is diminished in abnormal degree, although uremia may occur also when the amount of urine is abundant if the secretion contains a deficiency of toxic substances which are retained within the body. Uremia is accordingly the result of an intoxication, or, as it is now customary to designate it, an **auto-intoxication**, as the condition is dependent upon toxic substances generated by the body itself.

The *nature of the toxic substances* is not known. The so-called urinous substances have been thought of (urea and its decomposition-product—ammonium carbonate—kreatinin, potassium-salts, sodium chlorid), but of late there has been a tendency to adopt the view that the responsible factors may consist in *putrefactive alkaloids—ptomains.*

Although diseases of the kidneys constitute the most common cause of uremia, they are, however, not the sole cause. Diseases of the urinary passages, also, may be attended with uremia if they interfere with the elimination of urine. Among diseases of the kidneys particularly **acute** and **chronic interstitial nephritis** give rise to uremia most commonly. It is noteworthy that at times severe irritation of one kidney may through reflex influences cause suppression of the function of the other kidney, and thereby give rise to uremia. A similar condition has been observed in connection with **nephrectomy** and the presence of a **calculus in a ureter.** In some cases uremia results from obstruction by a calculus, not of one *ureter* merely, but of each. Occlusion of both ureters may be caused by new-growths of the bladder, the rectum, and the uterus, as well as by tuberculosis of both ureters. Among **diseases of the bladder** paralysis of this viscus, and in connection therewith urinary stasis, may induce uremia. Even when the bladder becomes over-distended in consequence of stupor there will be danger of uremia. This may develop also if the orifice of the bladder is obstructed by calculi, new-growths, or pressure from without. Finally, **strictures, laceration,** and **compression of the urethra,** and phimosis may be causes of uremia. Uremia may occur at any period of life. In children it develops most commonly as a result of acute nephritis following scarlet fever.

Symptoms and Diagnosis.—The symptoms of uremia consist particularly in *nervous* and *inflammatory manifestations*, the former being the result of irritation of the brain by the retained toxic substances, and the latter being due to the fact that the resistance of the tissues of the organism is diminished when the latter is saturated with excrementitious products. A most common symptom is **headache—cephalalgia**—which at times is diffused over the entire head, at other times is located in the forehead, the vertex, or the occiput, and at still other times exhibits a unilateral distribution—uremic hemicrania. Obstinate **neuralgia**, particularly supra-orbital and occipital, is not rarely dependent upon uremia. Some patients complain of migratory **muscular** and **articular pains. Vertigo, a sense of weight and pressure in the head**, also are symptoms of uremia. Frequently **disorders of the special senses** are present; the pupils not rarely are unusually small—*uremic myosis*—and they react but sluggishly to light. At times sudden blindness occurs—*uremic amaurosis*—which may disappear in the course of a few hours or days. Many patients complain from time to time of *deafness* and *tinnitus aurium*. An important symptom of uremia is frequent and persistent vomiting—**uremic hyperemesis**—probably in consequence of irritation of the vagus. **Consciousness** is often obtunded. The patients lie for days, and even for weeks, in a state of stupor, and are often delirious. At times they become violent and maniacal, and enter into a pronounced psychopathic condition. When consciousness is lost the **respiration** often acquires a Cheyne-Stokes character. **Uremic convulsions** occur frequently. These are usually clonic, and but rarely tonic. They may involve circumscribed groups of nerves and muscles, and consist in briefly transient muscular twitching; or there may be general clonic convulsions, with loss of consciousness, completely resembling an epileptic attack—*uremic eclampsia*. The duration and the number of the convulsions are susceptible of great variation. At times the attacks succeed one another at short intervals for days, the patient not returning to consciousness after the termination of the individual attack. At times they present the distinct character of cortical convulsions, beginning in a given extremity, and then, in accordance with the anatomic situation of the individual motor cortical centers, extending to the remaining extremity of the same side, and then to those of the opposite side.

Rarely *uremic paralysis* occurs, in the form of monoplegia and hemiplegia. Anatomic alterations may not be discernible in the central nervous system. In isolated cases I have observed circumscribed edema of the pia in the neighborhood of the central convolutions of the cerebral hemisphere on the side opposite to that of the hemiplegia. At times transitory paralysis persists after uremic convulsions. I have, however, also observed permanent hemiplegia in consequence of cerebral hemorrhage during the convulsive seizure. Sometimes violent *uremic tremor* is noticeable.

Some patients complain of **paresthesia** (crawling, pricking, a sense of cold). Particularly disagreeable is the often insatiable itching of the skin—**cutaneous pruritus.** Among the nervous manifestations of uremia are attacks of intense dyspnea—uremic asthma. Attacks of **uremic cardiac asthma** have also been observed. Among the *inflammatory manifestations* of uremia, diseases of the skin may be mentioned, and which have been dignified with the euphonious designation of *uremides.* Among these belongs particularly chronic **eczema,** although I have also observed herpes zoster in the course of uremia. At times obstinate **diarrhea** occurs. This may be characterized by the putrid odor of the stools, which contain at times necrotic shreds of the intestinal mucous membrane. Some patients suffer from obstinate **hoarseness,** the cause for which may be found on laryngoscopic examination to be chronic edema of the laryngeal mucous membrane.

In isolated rare instances I have observed yellowish [illegible] [illegible] upon the laryngeal and pharyngeal mucous membrane, from which superficial uremic ulcers developed.

A most serious manifestation is the occurrence of acute edema **of the glottis,** which may cause death by suffocation within a short time. At times symptoms of pneumonia appear suddenly. The serous membranes are particularly predisposed to inflammation, so that uremic **pleurisy** or **pericarditis,** less commonly **peritonitis** or **meningitis,** is encountered. The saturation of the body with toxic, excrementitious substances in uremia is at times indicated by symptoms of **blood-dissolution,** which can scarcely occur otherwise than in consequence of impairment of the nutrition of the walls of the blood-vessels, so that these readily rupture or become unduly permeable for red blood-corpuscles. Often frequent or profuse bleeding from the nose—uremic epistaxis—occurs; or there may be **vomiting of blood**—uremic hematemesis. Also **cutaneous hemorrhage**—uremic **purpura**—and bleeding from the gums have been observed. Uremic patients generally present a pallid **complexion.** The facial expression appears distorted, and the eyes often stare vacantly into distance. In many instances the expired air possesses a urinous odor, obviously resulting from decomposed urea, and thus from ammonium carbonate. In isolated instances of uremia following scarlet fever and cholera a whitish deposit has been observed to remain upon the skin after previous sweating, and which upon chemic examination proved to be urea—so-called **uridrosis.** The **bodily temperature** is subnormal in the majority of cases, although elevation of temperature may occur when inflammatory processes arise, at times with uremic convulsions, and at times also without demonstrable cause. Great attention should be given to the **state of the pulse**, for some cases of uremia depend upon attacks of cardiac failure, and this can be most certainly recognized from the smallness of the pulse and its deficiency in tension. At times the outbreak of an attack of uremia is preceded by unusual slowing of the pulse. The **urine** is generally diminished in amount. If its elimination can be augmented, the large number of tube-casts in the urinary sediment is often striking, and which obviously had previously obstructed the uriniferous tubules.

The *course of uremia* may be acute, subacute, or chronic. It may happen that apparently healthy persons are suddenly seized with uremic convulsions and death occurs. Perhaps examination of the urine after death will for the first disclose the fact that the patient has suffered from an unrecognized contracted kidney. The subacute cases may extend over eight weeks, and even more, while the chronic cases persist for months and years. In the latter event it is the rule for such marked improvement to occur at times that the patient believes himself cured. *Death* may take place in a uremic convulsion from cerebral or neural paralysis, or it may be due to edema of the glottis or of the lungs, or an attack of pneumonia, of pleurisy, or of pericarditis may terminate life. At times exhaustion becomes excessive.

The *diagnosis of uremia* is easy if adequate attention be directed to the eliminative relations of the urine and the presence therein of albumin. Otherwise confusion with diseases of the brain, epilepsy, meningitis, mental disease, cutaneous disease, disease of the stomach, and even other diseases, is scarcely to be avoided. Naturally a diagnosis of uremia will never be sufficient, but in every instance an effort must be made to determine the nature and the causes of the condition, because the treatment will be indicated accordingly.

Anatomic Alterations.—No specific anatomic conditions are as yet known to be associated with uremia. At times, but by no means with particular frequency, edema and anemia of the **brain** are observed. The inconstancy of these conditions is unequivocal testimony against the view that the nervous manifestations of uremia are dependent upon edema and anemia of the brain. At times peculiar necrotic alterations and destructive processes take place upon the **intestinal mucous membrane—uremic intestinal ulcers.** These have been attributed to irritation of the mucous membrane by ammonium carbonate resulting from the decomposition of urea excreted from the blood-vessels and deposited upon the mucous membrane.

Prognosis.—The prognosis of uremia is always serious, although it depends largely upon the fact whether the causes are susceptible of removal or not. Accordingly, it is, for instance, more favorable in cases of acute nephritis, or of impaction of a calculus, than in those of contracted kidney, because under the latter condition the disease is incurable.

Treatment.—The treatment of uremia must always be an individualizing and causal one. Phimosis or stricture of the urethra should be at once relieved. In the presence of stupor or of paralysis of the bladder the urine should be systematically evacuated with a carefully sterilized catheter. In order to increase the secretion of urine and hasten the elimination from the body of possible toxic products of metabolism an abundance of *milk* should be recommended. When stupor is present *intestinal infusions* of lukewarm milk or of physiologic salt-solution (0.75 per cent.) should be made thrice daily. If the patient does not retain the infusion, even though not more than 300 c.c. of fluid have been used, subcutaneous infusion of salt-solution should be practised. If the secretion of urine does not improve, *diuretics*, *diaphoretics*, or *drastics* may be employed to cause increased elimination of water. Should these measures also fail, resort should be had to *venesection*. If the patient exhales a markedly urinous odor, *lemonade* and *benzoic acid with camphor* should be given in order to neutralize the ammonium carbonate:

℞ Benzoic acid,	0.3 (4½ grains);
Camphor,	0.05 (¾ grain);
Sugar,	0.5 (7½ grains).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

Feebleness of pulse, faintness of heart-sounds, and other signs of cardiac weakness require the administration of large doses of *digitalis:*

℞ Powdered digitalis-leaves,	0.1 (1½ grains);
Diuretin,	1.0 (15 ");
Sugar,	0.5 (7½ ").—M.

Make 10 such powders.
Dose: 1 powder every three hours.

The last two indications can also be met by the following prescription:

℞ Powdered digitalis-leaves,	0.1 (1½ grains);
Benzoic acid,	0.3 (4½ ");
Camphor,	0.05 (¾ grain);
Sugar,	0.5 (7½ grains).—M.

Make 10 such powders.
Dose: 1 powder every three hours.

At times individual prominent symptoms will require **symptomatic treatment**. Attempts have been made to control persistent eclamptic convulsions by continuous *inhalation of chloroform*. For uremic neuralgia *phenacetin* or *antipyrin* may be advised (1.0—15 grains—thrice daily). Marked itching of the skin may be relieved by tepid baths (28° R.—35° C.—95° F.) and inunctions of the skin with a *carbolated ointment* (carbolic acid, 2.0—30 grains; wool-fat and lard, each, 25.0—¾ ounce), etc.

HYPOSTATIC KIDNEY (VENOUS HYPEREMIA OF THE KIDNEY).

Etiology.—Hypostatic kidney develops when the escape of venous blood from the kidney is interfered with. The condition develops most commonly in the sequence of general venous stasis in connection with **chronic diseases of the heart** and **the respiratory organs.** Enfeeblement of the right ventricle is invariably the source of the stasis in this connection. Less commonly hypostasis of the kidney develops in association with **diseases of the abdominal cavity,** as, for instance, abdominal tumors that exert pressure upon the inferior vena cava above the point of entrance of the **renal** veins. Finally, hypostatic kidney may develop as a result of **local obstruction** or **occlusion of the renal veins,** as, for instance, by thrombosis. Instances of the latter variety, which are as yet of little significance clinically, are distinguished from those of the two varieties first named by the fact that the arterial supply to the kidneys is unaltered, while under other conditions it is likewise interfered with.

Anatomic Alterations.—Hypostatic kidneys are generally enlarged. Accordingly the capsule of the kidney appears distended on section of the organ—retracts from the cut surface. The surface of the kidney appears smooth and presents a dark-red color. Frequently the greatly distended stellate veins upon it are conspicuous. Upon section of the kidney, although the cortex is deeply reddened and the Malpighian bodies often appear as dark-red granules, the reddish-black discoloration of the medullary structure is particularly marked. The consistency of the renal tissue appears increased, and this fact, in conjunction with the dark-red color, has given rise to the designation *cyanotic induration of the kidneys.*

Microscopic examination discloses great over-distention with blood of the glomerular loops and of all of the veins. If the condition has existed for a long time, however, striate thickening of the glomerular capsules and of the membrana propria of the uriniferous tubules takes place. There may be also hyperplasia of the interstitial connective tissue. As a result of contraction of the connective tissue the surface of the kidney acquires a nodular appearance, and often the capsule of the kidney is in places adherent to the surface of the organ. Such a condition is designated also *cyanotic contracted kidney.* At times extravasations of blood have taken place into the interstitial connective tissue, the Malpighian capsules, and the uriniferous tubules. At times, however, only the remains of hemorrhage are found, in the form of brownish granules of hemoglobin.

Symptoms and Diagnosis.—Hypostasis of the kidney is **attended with characteristic alterations in the urine.** The amount of urine is decreased (below 1500 c.c.), largely as a result of diminished arterial supply, for with occlusion of the renal veins alone the secretion of urine has rather been found increased. The urine is reddish in color (high-colored, saturated), its reaction is strongly acid, and its specific gravity is increased (usually above 1020). The urine contains albumin, but only in small amount, and the urinary sediment isolated hyaline tube-casts, round cells, and at times also a few red blood-corpuscles. Often urates are precipitated as a pulverulent deposit—so-called **brick-dust or lateritious sediment.** In addition there occur **edema of the lower extremities, ascites,** and **other manifestations of stasis.** In contradistinction from diffuse, acute nephritis, in the diagnosis of hypostasis of the kidney it is to be borne in mind that the disorder is generally induced by chronic disease of the heart or the respiratory organs, and that the amount of albumin and the number of tube-casts and of red blood-corpuscles is usually small. The *course of hypostasis of the kidneys* is, as a rule, chronic, in accordance with the causative conditions. Often exacerbations and remissions occur in conformity with the functional activity of the right ventricle. Hypostatic kidneys as such scarcely ever cause death, which, as a rule, results in consequence of cardiac paralysis or of suffocation from excessive hydrothorax and hydropericardium.

Prognosis.—The prognosis depends upon the curability of the causative conditions. Usually it is possible to increase the strength of the heart and to overcome the manifestations of stasis repeatedly. Subsequently, however, the remedies fail to exert their effect, and death is then unavoidable.

Treatment.—The treatment is the same as that for weakness of the myocardium (pp. 23 and 24).

DIFFUSE NEPHRITIS.

ACUTE DIFFUSE NEPHRITIS.

Etiology.—Acute diffuse nephritis is either of **infectious** or of **toxic** nature. *Infectious acute nephritis* occurs most commonly in connection with *infectious diseases.* It is encountered with particular frequency in the sequence of scarlet fever and diphtheria, but it may arise also in the sequence of other infectious diseases. Among the more frequent antecedent conditions may be mentioned fibrinous pneumonia, erysipelas, typhoid fever, acute articular rheumatism, malaria, syphilis, etc.

In relation to the connection between infectious diseases and acute nephritis, two possibilities may be considered: namely, either the conveyance of specific bacteria or pyogenic cocci to the kidneys, with the development of inflammation; or irritation of the kidneys in the elimination of bacterial poisons—toxins—through the kidneys. In accordance with previous experi- [illegible] possibility appears to be the more commonly effective.

At times acute nephritis occurs as an **independent infectious disease.** I have observed such occurrence repeatedly in members of a family and in servants in endemic distribution, and have demonstrated the presence of the Staphylococcus pyogenes albus in the urine. In the development of *refrigeratory* (*rheumatic*) and *traumatic acute nephritis* exposure to cold and injury are probably only contributing factors, favoring infection of the kidneys with bacteria, among which the Streptococcus pyogenes has been recognized in some cases. Among the varieties of *acute infectious nephritis* belong also those arising by *extension* from adjacent disease. Thus, acute nephritis not rarely complicates gonorrhea, cystitis, pyelitis, or paranephritis. *Toxic acute nephritis* may be induced by the use of certain medicaments and poisons, among which may be mentioned cantharides, oil of turpentine, other balsamics, active diuretics, potassium chlorate, potassium nitrate, quinin, sulphuric, nitric, hydrochloric, and carbolic acids, mercuric chlorid, etc. In addition to the digestive tract, the injurious substances may gain entrance into the body in the act of respiration (oil of turpentine) through the air-passages, or by means of inunctions and the like. Irritating ointments containing carbolic acid, pyrogallic acid, and remedies for scabies are well adapted to induce acute nephritis. Possibly that form of nephritis that at times develops in the sequence of *chronic cutaneous eruptions* and *burns of the skin* is a variety of toxic nephritis. Acute diffuse nephritis occurs at all periods of life. In children it develops generally in connection with diphtheria and scarlet fever. At times the primary disorders are so ill defined as not to attract attention, and the acute nephritis appears to be an independent disease.

Anatomic Alterations.—In the presence of acute, diffuse nephritis the kidneys are enlarged, and their capsules are therefore tensely distended, on section quickly retracting toward the hilus. The surface of the kidney is smooth and presents a greater or lesser number of small extravasations of blood. As a rule, the surface of the kidney is of a vivid, red color, although now and then pale kidneys are encountered. Upon section small extravasations of blood are appreciable, particularly in the cortex. At times the Malpighian bodies are conspicuous on account of increase in size, and marked distention with blood. The medulla of the kidney often presents a deep-red color.

On *microscopic examination of the kidneys* all varieties of acute nephritis probably agree in the distention with blood of the blood-vessels and the vascular loops. Extravasations of blood are frequently encountered, at times in the interstitial connective tissue, at other times in the capsule, and at still other times, finally, within the uriniferous tubules. The involvement of the Malpighian bodies, the interstitial connective tissue, and the epithelium of the uriniferous tubules, in the inflammatory process, is susceptible of wide variations. Often the cavity of the capsule is filled with an albuminous fluid, which has in part displaced the loops of blood-vessels from the inner surface of the capsule. At times concentric thickenings of the capsule, resembling the layers of an onion, are appreciable, greatly diminishing the cavity of the capsule, and at the same time compressing the vascular convolution. Cell-multiplication has, however, taken place also between the individual vascular loops. The nuclei of these loops are themselves increased in number, here and there in a state of fatty degeneration, and in places the vessels are occluded by thrombi., Under such circumstances the condition is designated *glomerulonephritis*, and these alterations are observed with particular frequency in connection with scarlet fever. More or less marked fatty degeneration has taken place in the *epithelial cells of the convoluted uriniferous tubules*. If the epithelial cells exhibit a marked tendency to desquamation, the condition has been described as *acute desquamative nephritis*. Frequently numerous uriniferous tubules are filled with tube-casts. Other tubules contain numerous round cells. Such a condition has also been described as *acute catarrhal nephritis*. The *interstitial connective tissue of the kidneys* contains collections of round cells, which at times may be so large as to be visible to the unaided eye as small nodules. This condition has been designated also *acute lymphomatous nephritis*. In the different varieties of acute diffuse nephritis at times one kind, and at other times other kinds of alterations preponderate. The question has been much disputed which of these alterations has been the earliest. In our opinion this is variable.

Symptoms and Diagnosis.—Acute diffuse nephritis, like most diseases of the kidney, is susceptible of recognition only from the **alterations in the urine**. Above all, the urine is conspicuous from its bloody appearance and the hemorrhagic sediment. At times the urine is light red in color, resembling meat-water, but frequently it is reddish brown or brownish black from the presence of methemoglobin in considerable amount. On microscopic examination of the urinary sediment red blood-corpuscles are generally found, presenting an unaltered appearance, or frequently a biconvex or a spherical shape. At times ameboid movement and constrictions are observable. Often the red cells have yielded up their coloring-matter, so that they appear as colorless discs of double contour—so-called blood-shadows. Frequently accumulations and collections of the smallest granules and flocculi of blood coloring-matter are observed. Cylindric structures also are not uncommonly present, apparently consisting of coherent hemoglobin. The urinary sediment often reveals the presence of blood by its brownish color. The amount of sediment is often quite considerable, so that it forms a dense cloudy precipitate. In addition to the red blood-corpuscles and hemoglobin, there occur in the sediment also round cells, epithelial cells from the uriniferous tubules, and tube-casts. The last are at times hyaline, at times granular, and are not rarely covered with round cells, epithelial cells from the uriniferous tubules, and red blood-corpuscles (Fig. 56).

Although the presence of blood in the urine—**hematuria**—is a common symptom of acute nephritis, it is not unexceptional, and cases occur in which the urine is free from blood. Under such conditions the detection of albumin in the urine—**albuminuria**—

and of tube-casts, round cells, and epithelium from the uriniferous tubules in the urinary sediment is of special importance. The amount of albumin in the urine may be so considerable that on boiling and addition of nitric acid the urine solidifies into a firm coagulum. The average amount of albumin is about 0.5 per cent. In addition the physical properties of the urine should be given consideration in cases of acute nephritis, as the **amount of urine** is diminished (below 1500 c.c.), its **color** deepened, and its specific gravity increased (about 1020). Naturally, hematuria cannot always be referred to acute nephritis. Renal hematuria is distinguished from **admixture of blood from the urinary passages** from the fact that the urine never contains clots of blood, but is intimately and uniformly admixed with blood. Renal hematuria occurs further in connection with **embolism of the renal artery**, although this develops generally in association with valvular lesions of the heart, and the disorder sets in suddenly with pain referred to the kidney. In cases of **hypostasis of the kidney** the urine contains little if any blood, little albumin, and few tube-casts, and, besides, the patients generally present chronic disease of the heart or the respiratory organs. In cases of renal hemorrhage from **carcinoma** and **tuberculosis of the kidney** the amount of albumin present in the urine is small, as is also the number of tube-casts, both at times even being wanting. **Traumatic hemorrhage from the kidneys** presupposes an antecedent injury.

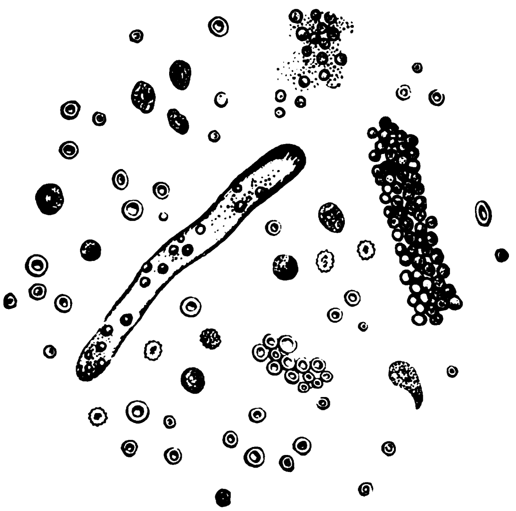

FIG. 56.—Urinary sediment from a case of acute nephritis following fibrinous pneumonia in a man, 42 years old; containing blood-casts, red blood-corpuscles, round cells, and epithelial cells from the uriniferous tubules and lower urinary passages; magnified 275 times (personal observation).

Local alterations in the kidneys are often wanting in cases of acute nephritis. Some patients, however, complain of a sense of pressure, of tension, and of pain in the region of the kidneys and in the sacral region. Bimanual palpation of the kidneys often fails to induce the slightest unpleasant effect. A frequent symptom of acute diffuse nephritis is **cutaneous edema.** This often involves the face earliest, or even alone, the eyelids particularly becoming swollen, and giving rise to remarkable narrowing of the palpebral fissure. At times marked edema of the entire skin is the earliest sign of which the patient has taken notice. This may reach so considerable a degree that the skin appears distended to the point of rupture. Often *accumulation of fluid* takes place *in the serous cavities*, and hydrothorax and ascites particularly are not uncommon complications. It is noteworthy that the patient generally exhibits marked **pallor,** and **examination of the blood** discloses that within a short time diminution in the number of red corpuscles and in the percentage of hemoglobin takes place. The **pressure in the aortic system** undergoes rapid change, and the blood-pressure increases. As a result the aortic-diastolic sound becomes accentuated, often with a tympanitic-ringing character, and the radial pulse becomes tense. In explanation of this manifestation irritation of the myocardium by metabolic products retained in the blood must be assumed. After a time *hypertrophy of the left ventricle* develops.

The increased arterial blood-pressure appears with especial distinctness in *pulse-tracings* (sphygmograms). While the reflux elevation becomes less, the first elevation due to elasticity becomes considerably increased. After complete recovery has taken place these manifestations are neutralized (Figs. 57 and 58).

The **bodily temperature** may remain unchanged, although as a rule it is elevated. At times the temperature reaches 39° C. (102.2° F.) and above, and if at the same time there should be abdominal distention, diarrhea, and enlargement of the spleen, confusion with typhoid fever is possible; but, naturally, the Widal blood-serum reaction will always be wanting in cases of acute nephritis. The patients complain frequently of drawing **pains in the muscles and the joints.** **Sleep** often is disturbed; while the

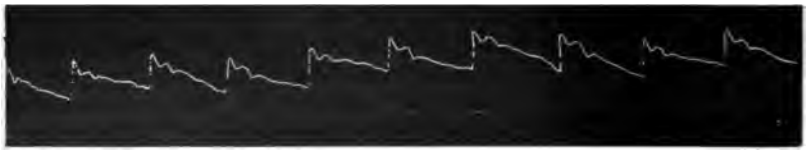

FIG. 57.—Pulse-tracing from a man, 22 years old, with acute refrigeratory nephritis, on the eighth day of the disease (personal observation, Zurich clinic).

appetite is lost, thirst is increased. Not rarely there are difficulty in the evacuation of urine, frequent vesical tenesmus, and pain in the act of micturition.

The *duration of acute nephritis* is susceptible of great variation. In cases of infectious disease—as, for instance, fibrinous pneumonia—acute nephritis is not rarely observed, disappearing within one or two days; and it is a remarkable fact that such speedy recovery may take place spontaneously although the primary disorder continues unchanged or is perhaps even progressive. In other instances acute nephritis may persist for from four to eight weeks. Cases also occur in which the disease persists for more than eight months, so that the condition can scarcely be designated *acute* nephritis. *Recovery* generally takes place in such a way that the hematuria becomes less pronounced and disappears entirely for a time; the albuminuria also slowly subsides, and likewise tube-casts and urinary sediment diminish, and finally the urine is free from all foreign elements. Often increased elimination of urine—polyuria—occurs with the onset of recovery, and the urine may have a low specific gravity (down to 1005). Not at all rarely it happens that exacerbations and remissions in the disease alternate with each other repeatedly.

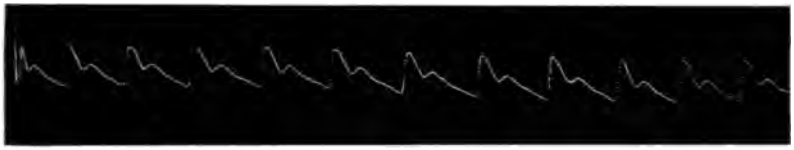

FIG. 58.—Pulse-tracing from the same patient (Fig. 57), after recovery had taken place, thirteen days later.

The *dangers of acute nephritis* consist especially in suffocation as a result of edema of the glottis or of the lungs, or of excessive effusions into the serous cavities, in inflammation of the skin and septicemia, in uremia, and in transformation into diffuse chronic nephritis. *Inflammation of the skin* may occur in consequence of marked cutaneous edema. The skin becomes reddened and ruptures at numerous points, so that edematous fluid escapes; at times erysipelas or gangrene of the skin develops, and, in consequence, absorption of the exciting agents of inflammation, general septicopyemia, which may terminate fatally.

Uremia frequently reveals itself earliest by headache and vomiting, until other uremic manifestations, particularly uremic convulsions, set in. The smaller the amount of urine eliminated, the greater is the danger of uremia. Experience has shown that the causes of acute nephritis also exert an influence upon the occurrence of uremia. Uremia is unfortunately common in connection with scarlatinal nephritis, while it scarcely ever attends diphtheric nephritis.

The *transformation of acute into acute chronic nephritis* is a rare occurrence. Under such circumstances the symptoms of acute chronic parenchymatous nephritis usually develop at first, with diminished secretion of dark urine of increased specific gravity, containing a large amount of albumin and fattily degenerated elements in the urinary sediment (fatty granules upon tube-casts, fatty granular cells, fattily degenerated epithelial cells), together with marked cutaneous edema. Gradually the symptoms of *secondary contracting kidney* may be superadded. Then the edema disappears, the urine becomes excessively abundant (above 2000 c.c.), with a low specific gravity, a small amount of albumin, and a scanty urinary deposit. Frequently death results from uremia.

Prognosis.—Acute nephritis is always a serious disease. No remedy with even an approximately reliable action is known, and, besides, uremia may set in at any moment and bring grave dangers. The possibility of transformation into chronic nephritis is but small, so that the prognosis is not materially made worse thereby.

Treatment.—No medicament capable of exerting a curative influence upon acute nephritis is as yet known. **Astringents** (also known as styptics or hemostatics) are without effect upon either the hematuria or the albuminuria, and also fuchsin, brombenzol, and strontium lactate, which have been recommended to diminish the elimination of albumin, are without any effect. Recovery from acute nephritis can therefore be brought about only through appropriate **dietetic measures.** The patient should remain constantly in **bed** as long as hematuria and albuminuria are demonstrable, for an equable temperature is always essential for patients suffering from disease of the kidneys, as also is bodily rest, for hematuria and albuminuria may be aggravated as a result of incautiously getting out of bed and walking around. A **tepid bath** at a temperature of 28° R. (35° C.—95° F.), morning and evening, is useful. The patient should remain in the bath for fifteen minutes, and is then dried with a warm towel, puts on a warm shirt, and returns to bed, which likewise has been previously warmed by hot-water bottles. Sudden cooling of the body is always markedly injurious for persons suffering from disease of the kidneys. The bath-room should therefore be so situated that the patient need not pass through cold apartments to enter the bath.

A **milk diet** is the best for the patient, as milk is both nourishing and easily digested, acts as a diuretic, and does not [illegible] the kidneys. Well-boiled milk should be preferred, as [illegible] and less readily undergoes fermentation in the [illegible] canal, and from one and a half to two quarts should be given daily, care being taken that the milk be swallowed slowly in small amounts at short intervals. Should the patient exhibit a distaste for milk, it may be mixed with weak tea or coffee or with beef-broth. All irritating articles of food should be avoided, as, for instance, strong coffee, strong tea, strong alcoholics, sharp condiments, vinegar, etc. Some meat, particularly white meat, may be permitted, and is without influence upon the excretion of proteids. Mashed potatoes and rice-pap, oatmeal-gruel with milk, and stewed fruit may be allowed. If symptoms of *uremia* appear, they should be treated according to the directions given on page 400.

In the presence of marked *cutaneous edema* **incisions** or **punctures** should be made **into the skin, if diaphoretic measures** (injections of pilocarpin, hot-air bath, hot bath) have not secured the desired result. If the edematous skin becomes inflamed, cataplasms containing mercuric chlorid (1.0 : 1000) or aluminum acetate (1.0 : 100) should be applied.

CHRONIC PARENCHYMATOUS NEPHRITIS.

Anatomic Alterations.—Chronic parenchymatous nephritis is designated also *large white kidney* on account of the appearance of the diseased organ. The kidney is considerably enlarged, and its capsule is tense and is readily stripped from the surface of the organ. The surface of the kidney is smooth, and presents a light butter-yellow color. Upon section of the kidney the renal cortex also presents a butter-yellow color, while the medullary structure is characterized by a red color. If the cut surface be scraped with the blade of a knife, a fatty deposit is made.

Microscopic examination of the kidney discloses particularly marked fatty degeneration of the epithelial cells of the convoluted uriniferous tubules, which are largely converted into fatty granular cells. Fatty degenerative processes occur also in the vascular loops of the Malpighian bodies, although in lesser degree. That the conditions present are inflammatory and not merely degenerative processes will be recognized from the fact that collections of round cells are present here and there in the interstitial connective tissue. The uriniferous tubules are often filled with tube-casts.

Etiology.—Little of a definite nature is known with regard to the causes of chronic parenchymatous nephritis. The condition is usually an *independent disease*, as cases in which the affection has developed from an antecedent acute nephritis are extremely uncommon. **Infectious and toxic influences** are the principal causative factors. Thus, the disease has been observed in the sequence of *malarial fever*, *pulmonary tuberculosis*, *syphilis*, *chronic suppuration*, *lead-poisoning*, *excessive indulgence in alcohol*. The patients are usually adults.

Symptoms, Diagnosis, and Prognosis.—Of all of the symptoms the **alterations in the urine** are the most important.

The urine is increased in amount, although on long-continued observation this may be found almost normal for days. The urine is dark reddish-yellow or yellowish-red in color, with an increased specific gravity (above 1020). The amount of albumin in the urine is often quite considerable (up to 5 per cent. and more). Generally the urinary sediment is abundant, and on microscopic examination it is found to be made up of tube-casts, round cells, and epithelial cells from the uriniferous tubules. Of particular importance is the presence of numerous fattily degenerated cells, fatty granular cells, as well as fat-drops upon tube-casts. The patients are conspicuous on account of **pallor of the skin**, and accordingly the **blood** is deficient in red corpuscles and hemoglobin. Marked cutaneous edema is present, and the face in consequence appears puffy and distorted. Also, the serous membranes are frequently filled with transudates. The disease generally begins insidiously, and often progressive pallor and cutaneous edema are the earliest conspicuous manifestations. Elevation of temperature does not occur except in the presence of complications. Increase in arterial blood-pressure takes place, although it is by no means so constant as in association with acute nephritis and contracted kidney. Most patients complain of loss of appetite and irregularity in the action of the bowels.

The *course of the disease* is generally chronic, and at times extends over several years. Occasionally *acute exacerbations* occur, in which the urine contains blood and entirely resembles that observed in acute nephritis. These exacerbations may in some cases occur with such frequency and persist for so long a time that the condition has been spoken of as *chronic hemorrhagic nephritis*. *Recovery* is almost impossible. Surgeons have reported isolated instances in which recovery took place after the removal of suppurating parts. In some cases *death* ensues from progressive *exhaustion*. In others it results from excessive edema, which may give rise to *asphyxia*, *cardiac paralysis*, *edema of the lungs or of the glottis*. It may also be induced by inflammation and gangrene of the skin, with consecutive *septicopyemia* secondary to marked edema of the skin.

At times chronic parenchymatous nephritis passes into *secondary contracted kidney* in consequence of hyperplasia of the interstitial connective tissue. Under such circumstances the amount of urine becomes increased, and may even exceed the daily amount passed in health. The color of the urine becomes light yellow, the specific gravity falls below 1015, and the amount of albumin in the urine diminishes until only traces can be detected. The amount of urinary sediment also becomes small, and the fattily degenerated cells become less and less numerous. The cutaneous edema disappears, while hypertrophy of the left ventricle progressively develops, with a hard, wiry pulse. The patients not rarely die in consequence of uremia, which is quite unusual in association with pure chronic parenchymatous nephritis.

Treatment.—The treatment for chronic parenchymatous nephritis is the same as that for acute nephritis (pp. 408 and 409). Nothing can be accomplished with drugs. The greatest importance should be attached to the **mode of life.** **Causal treatment** is indicated in cases of syphilis or lead-poisoning, and will then consist in the administration of potassium iodid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily). Suppuration should be controlled as speedily as possible, and if by no other means by removal of diseased parts.

CHRONIC INTERSTITIAL NEPHRITIS.

Anatomic Alterations.—Chronic interstitial nephritis is so designated from its chronic course, and is attended particularly with inflammatory hyperplasia of the interstitial connective tissue. As a rule, but by no means invariably, the hyperplastic connective tissue undergoes contraction after a time, as a result of which the kidneys are reduced in size. From this fact is derived the name *contracted kidney.* As the surface of the kidney is generally uneven and nodular, the condition is spoken of also as *granular atrophy of the kidneys. Several varieties of contracted kidney* must be distinguished accordingly as the process is *primary* or *secondary,* and, if primary, whether *juvenile* or *senile* (*arteriosclerotic*).

Some clinicians make a distinction further between *embolic contracted kidney* and *hypostatic contracted kidney.* In embolic contracted kidney connective-tissue hyperplasia occurs in the sequence of embolism of the renal artery. Should several emboli be present in the kidney, the organ acquires a multinodular, irregular surface and an atrophic appearance. The condition is almost always observed in association with valvular disease of the heart. It has already been pointed out that connective-tissue hyperplasia and contraction may occur in connection with hypostasis of the kidney. These two varieties are to be distinguished from *cardiac contracted kidney,* because in this condition the kidney is involved only secondarily in the sequence of disease of the heart, and the affection does not represent an independent disease of the kidney.

Primary or *genuine juvenile contracted kidney* occurs in individuals less than forty years old. The capsule of the kidney is often so firmly adherent to the surface of the organ that it can be removed only with some laceration of the kidney. The kidneys are conspicuous for their smallness, at times being reduced as much as one-third in size and weight. The surface of the kidney is multinodular, and the individual prominences may be of approximately the same size. These consist of relatively healthy renal tissue, while the depressions between them result from contracted connective tissue. Here and there cystic cavities of varying size, filled with water, at times with colloid fluid, are present. The surface of the kidney is grayish red in color. On section it will be found that the kidney cuts with peculiar resistance and almost like leather. The cut section, like the surface, is grayish red in color. The cortical tissue is diminished in amount, being in places contracted to a band one millimeter thick. The medullary substance, together with the calices of the kidney, is also diminished. The pelvis of the kidney appears rather dilated.

On *microscopic examination* the changes in the *interstitial connective tissue* are especially conspicuous. There will be found here foci of round-cell accumulation, which form preferably in the neighborhood of the Malpighian bodies, and the interstitial connective tissue besides appears increased, so that the uriniferous tubules are separated from one another by intervals of unusual size. The *Malpighian bodies* also participate in the connective-tissue hyperplasia, and their capsules are transformed into thick, striated, and cellular membranes. With progressive increase of connective tissue the capsular cavity becomes reduced, and finally complete connective-tissue obliteration of the capsule takes place. The *uriniferous tubules* do not remain uninvolved in the processes described. Often they are constricted off by connective-tissue hyperplasia, at times in garland-like arrangement in several divisions. Should fluid collect in the constricted portions, cystic cavities form that are appreciable microscopically. For this purpose it is naturally necessary that they shall have attained considerable dilatation. Only in recent years has attention been called to *changes in the blood-vessels*, which indicate that juvenile contracted kidney is in all probability of vascular origin. These consist in thickening of the intima of small arteries, which in places has led to closure of the lumen of the vessel—*chronic obliterating endarteritis*. In addition, thickening of the muscular layer and deposition of hyaline masses take place.

Senile or *arteriosclerotic contracted kidney* results from arteriosclerosis of the renal artery and its branches, and is therefore of vascular origin. It generally develops in advanced life, because arteriosclerosis is a disease of that period. Also, in cases of arteriosclerotic contracted kidney the capsule of the organ is often adherent to the surface of the kidney. The surface of the kidney appears uneven and nodular, and often exhibits cystic cavities. The color of the kidney is rather gray than red. On section of the kidney its tough consistency is less marked than in the juvenile contracted kidney. The cortex in places is greatly diminished. Microscopic examination discloses particularly that the interstitial connective-tissue hyperplasia is not uniform, but is focal in distribution, and that calcification is often present in the partially destroyed Malpighian bodies.

Secondary contracted kidney develops from chronic parenchymatous nephritis as a result of a gradual washing out with the urine of the fattily degenerated epithelial cells from the convoluted uriniferous tubules, so that the tubules undergo partial collapse, while interstitial connective-tissue hyperplasia preponderates. Microscopically the secondary contracted kidney is characterized by the preservation of a yellowish or yellowish-gray color, th yellow areas corresponding to fattily degenerated epithelial cells in the convoluted uriniferous tubules, and the gray areas to the hyperplastic interstitial connective tissue. At times the kidney acquires a yellowish, mottled appearance, so that it has been described as a *spotted* or *mottled contracted kidney.* Further, the secondary contracted kidney may be unchanged in size and possess a smooth surface, so that the interstitial connective-tissue hyperplasia will be disclosed only on microscopic examination.

Etiology.—Chronic interstitial nephritis results in consequence of **infectious, toxic,** or **involutional influences.** The *infectious varieties of contracted kidney* include those that develop in the train of infectious diseases, such as malaria and syphilis. Urocystitis and gonorrhea also at times cause contracted kidney. *Toxic contracted kidney* develops in consequence of the action of lead as the so-called *plumbic contracted kidney.* Disorders of metabolism, and particularly gout, may also be mentioned as causes. *Arteriosclerotic contracted kidney* is a result of involutional processes that attend advanced age, but nothing further is known with regard to the actual processes concerned. In cases of *secondary contracted kidney* the causes of the antecedent chronic parenchymatous nephritis must be taken into consideration, and these, as has been mentioned, are usually of infectious or toxic nature. Contracted kidney occurs with exceptional rarity in children, and it is then usually a secondary condition. Further, it is at times impossible to elicit a cause for the disease. Sometimes the disorder is **hereditary,** possibly because a morbid condition of weakness of the walls of the blood-vessels is inherited. **Exposure to cold** and to **wet,** and **indulgence in alcohol,** create a liability to the disease, and were considered by earlier clinicians as the actual causes.

Symptoms and Diagnosis.—Contracted kidney usually develops insidiously, and is unattended with cutaneous edema. It is therefore not uncommon for the disease to be discovered quite accidentally on examination of the urine. Naturally there are certain **suspicious symptoms** that should always stimulate an experienced and cautious clinician to subject the urine to careful examination. Among these may be mentioned palpitation of the heart, persistent headache, impairment of vision, repeated epistaxis, persistent hoarseness, frequent vomiting, obstinate eczema, and itching of the skin. The majority of these symptoms are of uremic nature. Also, the sudden occurrence of eclamptic convulsions or of cerebral hemorrhage is often dependent upon chronic interstitial nephritis.

The **urine** is increased in **amount,** and instead of from 1500 to 2000 c.c. the daily amount may reach from 3000 to 6000 c.c. and even more. Often the patients are aroused from sleep by the **frequent desire** to evacuate the bladder. The urine is pale yellow **in color,** and often exhibits a distinct tendency toward a greenish tint. Frequently the urine is slightly turbid, and, after shaking, the froth is retained upon its surface for a long time, a property that is often possessed by urine containing abnormal ingredients (albumin, sugar, biliary coloring-matter). The **specific gravity** of the urine is diminished (below 1015), at times as low as 1002. The **reaction** of the urine is feebly acid. Only a slight sediment is precipitated from the urine, and not rarely none at all, and this upon microscopic examination is found to contain hyaline and granular tube-casts of varying width. The amount of albumin present is always small, but albumin may also be absent for days and weeks. Under such conditions it is possible to confound the disease with **simple polyuria—diabetes insipidus**—for in this disorder also the amount of urine is increased and the specific gravity is diminished; but polyuria is unattended with the alterations in the heart and the pulse, and usually also with the retinal changes that attend contracted kidney. Confusion with **glycosuria—diabetes mellitus**—is scarcely possible, because in this condition the specific gravity of the urine, which is increased in amount, is abnormally high. At times **irritative states of the urinary passages**, as, for instance, catarrh of the urinary bladder, gonorrhea, tuberculosis, cause increased secretion of urine, with a reduction in specific gravity, but the urinary sediment will not contain tube-casts, if albumin be present its amount will correspond with the admixture of pus or of blood with the urine, and changes in the circulatory apparatus and in the retina do not occur.

The **alterations in the circulatory apparatus** take a conspicuous part in the clinical picture of contracted kidney. *Hypertrophy of the left ventricle* develops with great constancy in conjunction with a generally slight dilatation. The hypertrophy of the heart-muscle gives rise to a heaving and resistant apex-beat, and an accentuated, frequently tympanitic and ringing diastolic (second) aortic sound. Dilatation of the left ventricle gives rise to increased extent of the apex-beat, and to its displacement outside the left mammillary line and below the left fifth intercostal space. Further, after a time dilatation and hypertrophy of the right ventricle frequently also develop.

The *connection between contracted kidney and hypertrophy of the myocardium of the left side of the heart* has not yet been definitely established. The conditions found in acute nephritis indicate that also here irritative influences due to toxic substances retained in the blood must be thought of. Endarteritic alterations similar to those that occur in the contracted kidney itself have, however, also been observed in the blood-vessels, and it is conceivable that the hypertrophy of the heart-muscle is attributable to these. The earlier view that the hypertrophy of the heart-muscle is a result of destruction of numerous blood-vessels in the diseased kidneys has properly been abandoned, for hypertrophy of the heart does not develop even after amputation of both lower extremities, although a far more extensive vascular area is removed, and the blood-pressure in the aortic system is in consequence greatly increased.

The hypertrophy of the left side of the heart is appreciable to the patient as **distressing palpitation,** which usually occurs particularly after mental and physical excitement, and after the ingestion of stimulating fluids (coffee, tea, alcohol). Reference has already been made to this complaint as one of the suspicious symptoms of contracted kidney. The hypertrophy of the muscle of the left side of the heart is manifested further in unusual **hardness of the radial pulse,** in correspondence with the increased blood-pressure in the aortic system. The pulse often feels like an iron wire, whence it has been designated also the *wiry pulse.* A trained hand can frequently detect the presence of contracted kidney from feeling the radial pulse.

In the *pulse-tracing* or *sphygmogram* the abnormal increase in blood-pressure is exhibited in the small size of the reflux elevation, and the unusually marked character of the first elasticity-elevation (Fig. 59). Further, the blood-pressure in the radial artery has been measured by means of the sphygmomanometer of v. Basch, and it has been found to be as high as 244 mm. of mercury instead of 160.

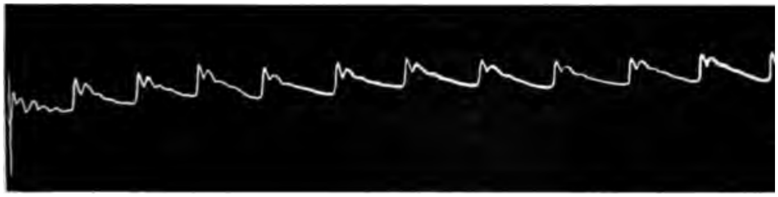

FIG. 59.—Pulse-tracing from the right radial artery in a case of chronic interstitial nephritis in a man 27 years old (personal observation, Zurich clinic).

Retinal changes occur, according to my experience, in one-fifth of all the cases. Particularly characteristic are yellowish spots around the macula lutea, which result from sclerosis of the axis-cylinders in the retina, and larger spots in the neighborhood of the optic papilla, which depend principally upon fatty degeneration of the granular layers. These alterations constitute the condition of *albuminuric retinitis.* In addition, there are frequently also swelling of the optic papilla and retinal hemorrhages, venous hyperemia, and tortuosity of the veins. The mode of origin of these nutritive disturbances in the retina is unknown. Visual acuity may be preserved in spite of the retinal changes, but frequently complaint is made of obscuration and impairment of vision.

Persons with contracted kidneys often present unusual **pallor,** which is dependent upon diminution in the number of red blood-corpuscles and in the percentage of hemoglobin. Rapid **emaciation** generally takes place, and the patients tire readily. The **skin usually** feels extremely dry, and exhibits a tendency to **desquamation.** Obstinate eczema also is frequently present. The **bodily temperature** exhibits no noteworthy alteration. Although the

appetite is impaired, the sense of **thirst** is increased, obviously in consequence of the increased elimination of urine, and often the patient complains of dryness of the mouth and of stickiness of the tongue.

The *course of contracted kidney* is chronic, and not rarely extends over several years. Most patients die of *uremia*, which may set in suddenly and may terminate fatally within a short time, or in other instances be repeated from time to time. Occasionally *cerebral hemorrhage—encephalorrhagia*—occurs in the first place, because in cases of contracted kidney the blood-vessels of the brain likewise often undergo degeneration, and also because hypertrophy of the muscle of the left side of the heart, and the resulting increase in the blood-pressure in the arteries, increase the liability to rupture of blood-vessels. At times symptoms of *weakness of the heart-muscle* appear. Gallop-rhythm becomes audible over the heart, hypostatic edema develops, and finally cardiac paralysis and asphyxia. Now and again *acute exacerbations* in the inflammatory process in the kidneys occur, and the urine contains blood and resembles that of acute nephritis. There is also a great tendency to *inflammatory processes in various organs*, and death not rarely results, particularly from pneumonia.

Prognosis.—Contracted kidney is an **incurable disease**, and the prognosis is accordingly grave. *Albuminuric retinitis* is an unfavorable symptom, for death usually follows within two years.

Treatment.—The treatment is the same as that for acute nephritis. Well-to-do patients may be recommended a sojourn in Southern climates in order to escape the dangers of fluctuating and cold weather. Particularly a residence on the borders of the African desert (Helouan) often has a remarkably favorable influence upon the albuminuria.

PURULENT NEPHRITIS.

Etiology.—Purulent inflammation of the kidney probably occurs only as a result of the entrance of pyogenic micro-organisms into the kidney. Chemic irritation and suppuration in the kidney scarcely occur in human beings. In addition to the Streptococcus pyogenes and the Staphylococcus pyogenes aureus and albus, the Bacterium coli particularly has been shown to be capable of exciting suppuration, and this organism appears to play the principal part in the etiology of suppuration of the kidneys. Pneumoniacocci and typhoid-bacilli have also been found in isolated instances. Bacteria can gain entrance into the kidneys in any of three ways: namely, through the *urinary passages*, the *blood-vessels*, or the *lymphatics*. *Infection of the kidneys by way of the urinary passages* is probably the most common, and just under such conditions the Bacterium coli appears to be the exciting agent with particular frequency. Gonorrhea and stricture of the urethra, cystitis, and pyelitis are not rarely attended with purulent nephritis. The disorder occurs with especial frequency in association with renal and vesical calculi. Urinary stasis of whatever origin greatly favors infection of the kidneys. Not rarely this develops in the sequence of surgical operations upon the bladder or the urethra—as, for instance, after the introduction of sounds or catheters—and the condition has been designated *surgical kidney*. Naturally the danger of infection is particularly great if most scrupulous disinfection of the instruments employed is not practised.

Infection of the kidneys by way of the arteries occurs in connection with *suppuration of the kidneys of embolic origin*. The suppuration associated with *ulcerative endocarditis* is best known, but similar processes are probably operative when purulent nephritis develops in the sequence of other *infectious diseases*. The *lymphatics* will constitute the medium of infection particularly when *inflammatory processes in the neighborhood* (paranephritis) have extended to the kidney. Infectious agents may be introduced into the kidney directly through wounds inflicted by instruments (dagger, knife, gun), but it should be borne in mind that suppuration of the kidney may occur also in consequence of closed injuries, as, for instance, a fall, a blow, or a contusion of the loin, and even after violent concussion of the body. At times it is impossible to elicit a cause for suppuration of the kidney—*cryptogenetic purulent nephritis*. Some patients then attribute the condition to cold, but on careful inquiry they are usually able to give no definite information with regard to this factor. In any event, *refrigeratory* (*rheumatic*) *suppuration of the kidney* can be accepted only in the sense that the exposure to cold alone did not excite the suppuration, but merely favored infection of the kidneys through alterations in the circulation and the resulting diminished powers of resistance of the tissues. Suppurative nephritis is an uncommon disease, which usually occurs after the fifteenth year of life, and is more common in men than in women.

Anatomic Alterations.—In advanced cases of purulent nephritis little or none of the tissue of the kidney will be left, so that the capsule of the organ, which frequently is thickened, consists of a sac filled with pus. Under such circumstances the condition may be designated also *pyonephrosis*, or, if the pelvis of the kidney has actively participated in the inflammatory process, *suppurative pyelonephritis*. In less advanced cases the kidney contains one or more small foci of suppuration, which here and there exhibit a tendency to coalesce. The suppuration generally begins in the form of small, scarcely visible sub.miliary and miliary abscesses, the coalescence of which gives rise to larger and larger accumulations of pus. In the presence of embolic abscesses of the kidney the infective embolus can be found at the center of the lesion. The cellular elements in the immediate vicinity of the embolus exhibit necrosis, and the peripheral boundaries of this necrotic area are surrounded by collections of round cells. Also, the uriniferous tubules are filled with bacteria, and not rarely form within the papillæ of the kidney straight grayish-white lines converging toward the apex of the papillæ. Purulent nephritis is at times bilateral, for both embolic processes and inflammatory processes arising by extension from the bladder are quite well capable of involving both kidneys in the morbid process.

Symptoms and Diagnosis.—In the diagnosis of purulent nephritis three groups of symptoms particularly are important, namely, those of general **septicopyemia, local alterations in the kidneys,** and **changes in the urine.** The disease, like all other suppurative processes, is attended with **fever,** which at times is interrupted by **chills.** The fever frequently exhibits a remitting type. There is a marked tendency to frequent, profuse, and debilitating **sweats.** The patients generally soon become pallid, lose appetite, complain of increased thirst, and not rarely suffer from diarrhea. The *local alterations in the kidneys* consist, in the first place, in *renal pain,* which may occur spontaneously or be induced by pressure in the loin. Frequently the diseased organ can be felt on bimanual palpation, and it will be found to be enlarged. At times *fluctuation* is appreciable.

As always in *palpating the abdominal organs,* the patient should lie with the head dependent and the lower extremities flexed at the hips and the knees, in order to relax the abdominal walls. The examination should be made only with warm hands, in order to avoid the rigidity of the abdominal muscles induced by cold. If necessary, the hands should be immersed in warm water. The patient should be engaged in conversation during the examination, in order to divert his attention therefrom. It may also be serviceable to have the patient lie upon the healthy side.

Naturally, foci of suppuration in the kidney must have attained a certain size before they can give rise to appreciable enlargement of the organ, and therefore such increase will not be present when the collections of pus are small. The associated pyelitis usually present may take a considerable share in causing enlargement of the kidney. It may also happen that the kidney is at times larger and at other times smaller, accordingly as the pus has free exit into the bladder or undergoes stagnation.

In the presence of purulent nephritis the **urine** contains pus—*pyuria*—as soon as the purulent accumulation has ruptured into the urinary passages. Under such conditions the presence of albumin can be demonstrated in the urine, to an amount corresponding with the amount of pus. Larger amounts of alb-

and tube-casts will be present in the urine only when a diffuse inflammation of the kidney has been superadded to the purulent nephritis. Blood is not rarely present in the urine if blood-vessels are perforated in the process of destruction of the kidney. The presence of desquamated renal tissue—*renal sequestra*—is particularly significant; but this occurrence is uncommon. The sequestra may at times attain the size of a pigeon's egg, and on microscopic examination exhibit the structure of renal tissue. Often the amount of urine is increased in consequence of reflex irritation of the renal nerves. Under such circumstances the urine is light yellow in color and of a low specific gravity. The urine often undergoes alkaline fermentation. Not rarely the patients complain of vesical tenesmus and of pain in the evacuation of urine.

The *diagnosis of suppuration of the kidney* may be extremely difficult, because in the presence of small encapsulated foci of pus in the kidney local alterations in the kidney and in the urine may be absent. Should pyuria be present, the possibility of **tuberculosis of the urinary organs** should be considered, although this would be attended with the presence of tubercle-bacilli in the urine. Purulent nephritis is distinguished from **suppuration in the urinary passages** by means of the local alterations in the kidney.

The *course of suppuration of the kidney* may be acute, subacute, or chronic, and in the last-named event it may extend over many months. Among the *complications renal colic* should first be mentioned. This results in consequence of temporary obstruction of the ureter by plugs of pus or exfoliated renal tissue, with the development of acute urinary stasis in the affected ureter and pelvis of the kidney. The patients then usually complain of sudden, severe pain in one loin, are at times seized with a chill and vomiting, and the bodily temperature rises to a higher level. The urine becomes diminished in amount and often quite clear, because it is secreted by the free healthy kidney. In consequence of acute urinary stasis in the pelvis of the kidney—*acute hydronephrosis*—the diseased organ either becomes palpable, or, if it has previously been palpable, it becomes increased in size. As soon as the obstruction to the flow of urine is removed the pain ceases, the temperature declines, and often remarkably large amounts of urine are evacuated, the fluid being now again purulent and turbid. Should the obstruction not be removable, there will be danger of *uremia* and *urinary septicemia.*

Among the less common occurrences are paralysis of the lower extremities—*urinary paraplegia*—which has been attributed to neuritis resulting by extension from the kidney.

The situation becomes extremely grave when the accumulation of pus ruptures not into the urinary passages, but into the sur-
uding tissues. *Rupture into the abdominal cavity* usually causes
tal peritonitis; rupture into the pararenal cellular tissue

gives rise to paranephritis. Rupture may take place also into the stomach, the intestine, the pleural cavity, or, after the previous formation of pleuritic adhesions, into the bronchial tubes. At times the pus ruptures externally, and an external renal fistula is formed. Under such conditions extensive burrowing of the pus is possible. Long-continued suppuration of the kidney at times gives rise to *amyloid disease* and its dangers. At times death results in consequence of *progressive exhaustion* or of *urinary septicemia*, the latter resulting from absorption of bacteria from the decomposed urine into the blood.

Prognosis.—The prognosis of purulent nephritis has become more favorable within recent years from an appreciation of the fact that the disease is a surgical disorder which cannot be influenced by internal remedies. Nevertheless the disease is still a serious one.

Treatment.—The single possible means by which an accumulation of pus in the kidney can be removed consists in **nephrotomy or nephrectomy.** Nephrotomy will suffice when the diseased organ still contains considerable healthy tissue. Otherwise nephrectomy should be performed, and the diseased kidney removed from the body.

EMBOLIC INFARCTION OF THE KIDNEY.

Etiology.—Embolic infarction of the kidney occurs principally in connection with **disease of the aortic** or **mitral valve,** in consequence of the detachment of small thrombotic vegetations that gain entrance with the blood-current into the renal artery, or more commonly into small branches thereof, and there become impacted. Only rarely is the condition dependent upon detached echinococcus-vesicles, or fragments of tumors of the myocardium, upon detached thrombi from arteriosclerotic areas in the aorta or from aortic aneurysms, or upon thrombi from the pulmonary veins.

Anatomic Alterations.—Emboli gain entrance most commonly into the blood-current through the *left renal artery,* because this arises from the abdominal aorta at less nearly a right angle than the right renal artery. Only rarely does an embolus remain lodged in the trunk of the renal artery. When it does, **necrosis** occurs in almost the entire portion of the kidney cut off from its blood-supply. As a rule, emboli are swept into the smaller branches of the renal artery, where they give rise to the formation of **wedge-shaped renal infarcts.** These reach the surface of the kidney with a broad base, while their apices are directed toward the medullary aspect. The wedge-shaped area cut off from its blood-supply undergoes speedy coagulation-necrosis, and acquires a yellowish-gray color. Its boundaries appear blood-stained from the presence of emigrated red blood-corpuscles. Subsequently fatty degeneration and absorption of the area may take place, so that only a small, superficial, depressed cicatrix remains upon the surface of the kidney. If the kidney is the seat of multiple emboli, it may acquire a multinodular appearance suggestive of contracted kidney, and the condition has been designated *arteriosclerotic contracted kidney.* At times calcification takes place in renal infarcts. Not rarely both kidneys are the seat of emboli.

Symptoms and Diagnosis.—Embolism of the kidney may be recognized generally from the sudden occurrence of **pain in the loin.** Pressure in the same situation discloses also tenderness. A **chill, followed by elevation of temperature and vomiting,** often occurs in consequence of reflex irritation. The urine becomes bloody—**hematuria**—and not rarely contains also tube-casts and rather more albumin than corresponds to the amount of blood. In addition the demonstration of a source of embolism, thus generally a valvular lesion of the heart, will be important in the diagnosis. The symptoms may disappear in the course of a few days, although recurrences are possible at any moment.

Prognosis.—Embolic infarction of the kidney is not a serious disorder, providing the embolus does not contain bacteria nor possess infectious properties, so that suppuration of the kidney is not to be feared.

Treatment.—**Rest in bed** and a **milk-diet** should be prescribed, an **ice-bag** applied to the affected loin, and if the pain be intense **a subcutaneous injection of morphin** may be given.

AMYLOID KIDNEY.

Etiology.—The causes of amyloid kidney are the same as those of amyloid disease of other organs—chronic wasting discharges and cachectic conditions of all kinds; as examples may be mentioned suppuration of bones and joints, chronic diarrhea, pulmonary tuberculosis, syphilis, malaria, and carcinoma. Only rarely does amyloid kidney develop without appreciable cause. As the same causative factors are also capable of inducing chronic parenchymatous nephritis, it will be understood that both diseases occur together with great frequency.

Anatomic Alterations.—*Uncomplicated amyloid kidney* is extremely rare. In the presence of diffuse amyloid degeneration the kidneys present a pale-yellow or whitish appearance, whence the designation waxy kidney. The kidneys are increased in size. Their capsule is readily detached from the surface of the organ, and the latter presents a smooth appearance. The consistency of the organ is increased. The renal tissue at times presents an appearance as if frozen, so that it can be shaved from sections of the dney. Thin sections appear translucent in transmitted light.

If a solution of iodin and potassium iodid be poured upon the cut surface, this will assume a mahogany-brown tint. In the majority of cases the large white kidney already described will be encountered, and the presence in this of areas of amyloid degeneration can be demonstrated from the appearance of the mahogany-brown color on addition of the solution of iodin and potassium iodid.

On microscopic examination the amyloid tissue is characterized by its waxy luster and its swollen and structureless appearance. It yields, besides, the amyloid reactions with the solution of iodin and potassium iodid, with the solution of iodin and dilute sulphuric acid and methyl-violet described on pp. 343 and 344. The vascular loops in the Malpighian bodies are attacked earliest; then the efferent vessels and the straight vessels are involved; and finally also the capillaries between the uriniferous tubules and in the medulla of the kidney, the capsules of the Malpighian bodies themselves, the membrana propria, and the epithelial cells of the uriniferous tubules.

Generally other organs also are the seat of amyloid degeneration, particularly the spleen, the cortex of the adrenal bodies, the liver, and the intestines.

Symptoms and Diagnosis.—In the presence of *uncomplicated amyloid kidney* the amount of urine is normal or slightly increased. The urine is pale yellow in color, and its specific gravity is diminished (from 1010 to 1015). The urine generally contains considerable albumin (up to 20 grams—300 grains—daily), although isolated instances are on record in which albuminuria was absent throughout. The urinary sediment is generally slight, and at times contains remarkably wide cylinders of waxy luster. Some tube-casts may also yield the amyloid reaction, although this is not distinctive of amyloid disease of the kidneys, as it may occur also in connection with diffuse nephritis. As a rule, there is marked **edema**. The primary disorder itself will cause **pallor of the skin**. Death usually takes place in consequence of progressive exhaustion or of excessive edema. Should amyloid disease of the kidneys exist in association with chronic parenchymatous nephritis, the symptoms of the latter will be the more conspicuous, and there is no infallible symptom by which the amyloid degeneration can be recognized with any degree of certainty. Amyloid kidney will be present, in all probability, if increased size and consistency of the spleen and the liver indicate amyloid degeneration of these organs. Should the intestine be involved in amyloid disease, diarrhea also will be present.

Prognosis.—The prognosis of amyloid disease of the kidney is unfavorable, as recovery appears impossible.

Treatment. In the first place, the same dietetic regulations should be prescribed as in diffuse nephritis (pp. 408 and 409). Among drugs iron and iodin have been employed, as, for instance:

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CARCINOMA OF THE KIDNEY.

Etiology.—Carcinoma of the kidney may be either *primary* or *secondary*. Often no cause is ascertainable for *primary carcinoma of the kidney*. The patients often attribute the condition to **traumatism**. Possibly **renal calculi** also may give rise to carcinoma of the kidney. Experience has shown that *men* are attacked oftener than women. Although carcinoma of the kidney occurs with particular frequency after the fiftieth year of life, it constitutes an exception as compared with carcinoma in other organs from the fact that it has been observed in numerous instances also in *children* within the first five years of life. *Secondary carcinoma of the kidney* results either through direct extension of the new-growth from the neighborhood, or through metastatic dissemination from remotely situated organs. Not rarely it develops in the sequence of carcinoma of the testicle, even if the diseased organ was removed many years previously.

Anatomic Alterations.—Primary carcinoma of the kidney generally develops only in one kidney, whereas secondary carcinoma frequently appears in both organs. In cases of secondary carcinoma of the kidney the new-growths generally consist in circumscribed nodules, whereas in cases of primary carcinoma diffuse infiltration of the kidney takes place. Under these conditions the kidney may be greatly increased in size and weight, although it usually retains its shape in considerable degree. On section of the kidney the new-growth will at times present a dense fibrous appearance, with a deficiency of fluid, at other times a soft and medullary appearance, and at times be traversed by cavities containing colloid material, so that a distinction is commonly made between *fibrous carcinoma* (*scirrhus*), *medullary carcinoma*, and *colloid carcinoma* (*alveolar* or *gelatinous carcinoma*). The neoplastic tissue is generally grayish white or yellowish white in color. At times it contains numerous blood-vessels, and then presents a reddish appearance. Also, blood-vessels may rupture and copious extravasations of blood take place into the carcinomatous tissue, and these, after having existed for a considerable time, acquire a reddish-brown, chocolate-like appearance. The carcinomatous cells are considered as offspring of the epithelial cells of the uriniferous tubules. Extensive carcinomata of the kidney not rarely **cause displacement of adjacent organs** (liver, stomach, spleen, intestine).

Symptoms and Diagnosis.—Carcinoma of the kidney may remain unrecognized so long as it does not undergo disintegration, and thereby give rise to *alterations in the urine*, or so long as demonstrable *enlargement of the kidney* is not present. Under the most favorable circumstances *latent carcinoma* may then be suspected from progressive emaciation and pallor, although doubt may remain as to the seat of the disease. Carcinoma of the kidney frequently gives rise to **hematuria,** resulting from perforation of blood-vessels in the process of disintegration of the carcinomatous tissue. Hematuria in advanced life, occurring without demonstrable cause, should always arouse suspicion of carcinoma of the kidney. The hemorrhage may be abundant, persist for a long time, be repeated at short intervals, and as a result cause alarming anemia and serious loss of strength. *Examination of the urinary sediment* may prove of importance, for, although it is true that carcinoma-cells present in themselves nothing peculiar, it will nevertheless always be highly suspicious if many cells are present, and particularly cell-groups with multiple nuclei. The bloody urine is often evacuated without discomfort. In some cases, however, attacks of **renal colic** occur which result from temporary obstruction of the ureter by blood-clots of considerable size, or by aggregations of cells. The patients often complain persistently of severe **pain in the loin,** which is particularly prone to occur at night and disturb sleep. At times the patients are able to walk only with the **body bent forward,** at the same time avoiding with fear all possible rotation or flexion of the vertebral column. **Pressure in the loin** is generally painful. If the kidney has become increased in size in consequence of carcinomatous proliferation, it at times gives rise to a considerable visible enlargement in the loin and of the abdomen. In diagnosis the detection on palpation of a **tumor in the loin,** which is usually nodular, painful, and hard, is important, although carcinoma of the kidney also may be fluctuating. If the carcinoma is of small size, it may at times be possible to reach it with the fingers by having the patient lie upon the healthy side of the body and make deep inspirations while bimanual palpation of the kidney is practised.

It is by no means always easy to determine with certainty that an abdominal tumor is of renal origin. It should be noted, in the first place, that the tumor in question is situated in the loin or has arisen therefrom. Besides, renal tumors do not undergo displacement with the respiratory movements, and generally retain the bean-shaped form of the kidney. They are separated from the lower border of the liver by a tympanitic zone on percussion, because the transverse colon passes between the two organs Finally, a sausage-shaped body usually passes over its anterior surface from below upward, and this may from time to time be seen to be the seat of peristaltic movement. This corresponds with the ascending or descending colon displaced from its normal situation. It becomes especially distinct on insufflation of the rectum with air, and on percussion it then yields a tympanitic note.

Extensive tumors of the kidney displace the liver, the diaphragm, the lungs, and the heart upward, and as a result cause marked **dyspnea, palpitation of the heart,** and a **sense of constriction.** Pressure upon the stomach and the intestines induces **vomiting** and irregularity in bowel-movement. Patients v cinoma of the kidney usually undergo rapid emaciation and become exceedingly pale. Death results, as a rule, within a year in consequence of *carcinomatous marasmus*. At times *complications* arise, such as *rupture of the new-growth* into the abdominal cavity, into blood-vessels, or externally. Rupture into the abdominal cavity is generally followed by rapidly fatal peritonitis, while rupture externally gives rise to an external renal fistula, and rupture into the blood-vessels to hypostatic edema. *Pressure upon adjacent nerves* may give rise to intercostal neuralgia or paralysis of one lower extremity. The tumor may also penetrate the vertebral canal through the intervertebral foramina, and cause *spinal pressure-paralysis*.

Prognosis.—The prognosis of carcinoma of the kidney, like that of carcinoma in other organs, is unfavorable. The only hope for recovery depends upon early operation.

Treatment.—Nothing can be accomplished in the treatment of carcinoma of the kidney by means of internal remedies. When the diagnosis has been established no time should be lost in the performance of **nephrectomy**. If the appropriate time for operation has been neglected, or if the patient declines operation, all that remains is symptomatically to administer **narcotics** for the relief of severe pain, and **styptics** for the control of profuse hemorrhage. In addition, an effort should be made to sustain the strength by means of a **nutritious diet**.

In addition to carcinoma, *sarcoma* and *adenoma of the kidney* possess clinical significance. Both of these are indistinguishable from carcinoma during life. Sarcoma of the kidney is frequently of secondary origin and bilateral. Sarcoma of the kidney is observed also in children in the first five years of life. At times sarcomata contain transversely striated muscular fibers (striocellular myosarcoma). Adenoma of the kidney not rarely originates from displaced elements of the adrenal gland.

CYSTIC KIDNEY.

Anatomic Alterations.—Cystic kidney consists in a transformation of one or often both kidneys into a multilocular structure, which may attain considerable proportions and greatly displace adjacent organs. The individual cavities frequently contain fluid, at times also colloid material in which, remarkably, no urea, and at most uric acid, can be demonstrated. At times scarcely a trace of renal tissue can be recognized. The walls of the individual cavities consist of connective tissue. Their inner surface is lined with longitudinally arranged endothelial cells; here and there collections of epithelial cells are present. The condition is probably always a *congenital* one, and it is often associated with other malformations (harelip, club-foot, absence of the renal pelvis ter, absence of the duct of Botal).

Opinions are divided as to the *mode of origin of congenital cystic kidney.* According to the view of some, it results from *urinary stasis during fetal life,* which may occur either in consequence of inflammatory alterations in the papillæ of the kidney and obstruction of the uriniferous tubules, or from failure in the establishment of proper relations between the glomeruli and the convoluted uriniferous tubules and the deeper uriniferous tubules in the development of the kidney. Others consider the condition as a *neoplastic one* (*cystadenoma*). Both views are supported by certain evidence: the first, for instance, by the fact that congenital cystic kidney has been observed in association with congenital phimosis, and the second by the fact that congenital cystic kidney may suddenly increase in size in advanced life. Possibly both modes of development occur.

Etiology.—Nothing is known with regard to the causes of congenital cystic kidney. At times several members of the same family are victims of the disease.

Symptoms and Diagnosis.—At times cystic kidneys attain such considerable proportions during fetal life as to cause *obstruction to labor,* and delivery can be effected only after embryotomy. In other instances the children are born alive, but present so large a *renal tumor* that death soon occurs from asphyxia and cardiac paralysis. In still other instances no symptoms at all are present for years. Then, however, the kidney becomes greatly enlarged, and gives rise to symptoms and dangers in consequence. Such observations naturally often give the impression that the disorder has developed after birth. In the diagnosis the demonstration of a **tumor of the kidney** is important in the first place, and this has been discussed on p. 424. The tumor itself often presents a nodular surface, so that it may be readily mistaken for carcinoma of the kidney. In contradistinction from abscess and echinococcus of the kidney and hydronephrosis, fluctuation is, as a rule, not elicitable. Should exploratory puncture be decided upon, it is important to bear in mind that urea will not be found in the fluid. The **urine** is frequently unaltered, although hematuria may occur periodically. Albuminuria is not rarely observed. The patients complain principally of a **sense of tension and of pain in the loin and in the abdominal cavity.** Displacement upward of the liver, the spleen, the diaphragm, the lungs, and the heart gives rise to dyspnea and palpitation of the heart, and at times causes *death by suffocation* or *paralysis of the heart.* Some patients die from *uremia* in consequence of suppression of urine. Cystic kidneys may also rupture, and thereby occasion serious dangers, as, for instance, perforative peritonitis.

Prognosis.—The prognosis of cystic kidney is unfavorable, because the cysts have a tendency to undergo enlargement, and in consequence to be attended with various dangers.

Treatment.—The treatment of cystic kidney is surgical, and consists in **puncture of the cysts** or **nephrectomy,** although the latter formed only when the other kidney is healthy with

ECHINOCOCCUS OF THE KIDNEY.

Etiology.—With regard to the cause of echinococcus of the kidney, the statements made upon pp. 350 and 351 concerning echinococcus of the liver are applicable. Human beings acquire echinococci by swallowing the ova of the tapeworm of the dog—Tænia echinococcus. An explanation is wanting for the fact that echinococcus-cysts develop at times in one and at other times in another organ.

Anatomic Alterations.—Echinococcus of the kidney is generally unilateral, and, as experience has shown, it is most commonly situated upon the *left side*. The cyst is *unilocular*, and it may at times be larger than an adult head, and, in addition to fluid, contain also daughter-cysts and granddaughter-cysts. Cholesterin-plates and crystals of uric acid, calcium oxalate, and ammonio-magnesium phosphate may frequently be demonstrated in the fluid. At times only small remnants of renal tissue are still present. The healthy kidney often appears hypertrophied.

Symptoms and Diagnosis.—The diagnosis of echinococcus of the kidney is absolutely certain only when **echinococcus-vesicles, echinococcus-scolices,** or **echinococcus-hooklets** can be demonstrated in the **urine**. This will be possible only when the mother-cyst has ruptured and evacuated its contents into the pelvis of the kidney. Under such conditions symptoms of renal colic (sudden pain, chill, fever, vomiting) frequently first appear, as long as the echinococcus-cyst causes obstruction of the ureter. Additional urinary disturbances occur when the echinococcus-cyst obstructs the urethra. At times the echinococcus-cyst is suddenly expelled forcibly for a considerable distance with a loud sound, and is succeeded by turbid, milky urine resembling soapsuds. Expulsion of echinococcus-vesicles with the urine may be repeated at intervals of varying length, and for a period of years. In this way natural recovery may take place. Should expulsion of echinococcus-cysts with the urine not take place, echinococcus of the kidney can be recognized only when a **tumor of the kidney** has developed. Should this be wanting, the cyst will remain concealed. The tumor of the kidney, the essential points in whose diagnosis have been stated on p. 424, is generally nodular and yields fluctuation, though not constantly. The differentiation from **cystic kidney, abscess of the kidney,** and **hydronephrosis** is often impossible. *Exploratory puncture* may yield the desired information from the discovery of echinococcus-scolices or echinococcus-hooklets in the fluid. At times an echinococcus-cyst of the kidney undergoes **suppuration**, and the clinical picture may then even more closely simulate that of abscess of the kidney. *Rupture of an echinococcus of the kidney* into adjacent organs (stomach, intestine, pleural cavity, lungs, and bronchi), or into the abdominal cavity, or externally, may also take place at times. When the kidney has undergone considerable increase in size **symptoms of pressure upon adjacent organs** will appear—gastric and intestinal disturbances, dyspnea to the point of asphyxia, palpitation of the heart, and cardiac paralysis.

Prognosis.—The prognosis of echinococcus of the kidney is not unfavorable if the disorder is looked upon as a surgical one, and too much time is not consumed with internal treatment.

Treatment.—Echinococcus-cysts cannot be destroyed by internal remedies. Injections of **mercuric chlorid** (1 : 1000) into the echinococcus-cyst may possibly cause death and contraction. In the presence of an extensive echinococcus-cyst of the kidney, **nephrotomy or nephrectomy** should always be resorted to, accordingly as considerable healthy renal tissue is present or not.

MOVABLE KIDNEY.

Etiology.—Movable kidney occurs with great frequency, particularly in *pallid* and *emaciated women*. Not rarely the kidney is forcibly displaced from its natural situation by **pressure,** particularly as a result of tight lacing, the wearing of constricting waist-bands, and the like. In men who from vanity wear corsets or tight belts movable kidney develops in the same way as in women. At times movable kidney is induced by **heavy lifting** and **persistent expulsive effort,** because the diaphragm as a result thereof exerts pressure upon the kidney. Wandering kidney is, therefore, common in persons with chronic cough and constipation. In some cases the condition has been observed to develop in the sequence of a **fall** or a **blow** upon or an **injury** to the **loin.** At times wandering kidney occurs as a result of **emaciation,** as, for instance, after protracted febrile disease, or after reduction-cures, obviously because as a result the capsule of the kidney likewise yields up some of its fat and its support of the kidney is lost. Occasionally wandering kidney appears after the **puerperium,** in consequence of relaxation of the abdominal walls and diminution in the intra-abdominal pressure. **Increased weight of the kidney** may also give rise to wandering kidney, as, for instance, when the organ is the seat of carcinoma or tuberculosis. At times the kidney is forcibly displaced from its situation, as, for instance, by **tumors of the liver,** the **spleen,** the **pancreas,** or in consequence of **kyphoscoliosis** of the vertebral column. In some cases **nervous influences** appear to be operative. Thus, movable kidney is not at all uncommon in cases of tabes dorsalis. The frequent association of wandering kidney and diseases of the female generative apparatus is likewise often referred to the nervous influence of the latter upon the kidneys. There is, probably, also, a *congenital predisposition* to wandering kidney. The affection is rare in childhood, although isolated instances of congenital movable kidney are on record.

Symptoms, Diagnosis, and Anatomic Alterations.—Many persons possess a movable kidney without suspecting the fact. The condition may be found accidentally on careful examination of the kidneys. It can be detected with especial ease if, with the abdominal walls relaxed, *bimanual palpation of the kidneys* is practised and *deep inspirations* are encouraged. Most commonly the **right kidney** is movable, and this is due to the fact that it is subjected to a certain degree of pressure by the superimposed liver. The left kidney is much less commonly affected, and least commonly both kidneys are movable. The mobility of the kidney may attain varying degrees of intensity. In the mildest cases the lower extremity of the kidney can just be felt on deep inspiration, and some clinicians even believe that this is a natural and not a morbid manifestation. In advanced cases the entire kidney can be felt, and be moved up and down in its fatty capsule. In the aggravated cases the kidney is wholly displaced from its normal situation, and may be found in the abdominal cavity, sometimes in the iliac fossa and even in the true pelvis. Some clinicians designate only cases of the last kind as *wandering kidney* or *migrating kidney*. Under such circumstances the kidney may possess such a degree of mobility that it can be readily moved to and fro in the abdominal cavity. Naturally it may happen also that the organ becomes adherent in some abnormal situation, where it again becomes immovable.

As a matter of course, marked downward displacement of the kidney is possible only when the organ possesses an abnormally long mesonephron. At the same time the renal artery and vein must be unduly lengthened. At times the patients themselves first notice a movable body in the abdomen accidentally, and come to the physician in great alarm, fearing the presence of carcinoma. Under such circumstances it often requires most earnest assurances in order to relieve them of this fear. In other instances the physician discovers the readily movable kidney accidentally in the course of an abdominal examination for some other purpose. That the body is the kidney will be recognized from the bean-shaped form of the tumor. Its surface is smooth, and pressure causes a dull, painful sensation. At times —according to my experience quite rarely—the loin upon one side will be found upon inspection to be depressed, and, on comparison with the opposite side, it will yield a tympanitic note on percussion, which will be replaced by a dull percussion-note when the movable kidney has been restored to its normal situation.

At times *pulsation of the renal artery* can be felt in the hilus of the kidney.

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Generally the condition corrects itself in the course of a few days. At times it disappears rapidly if the kidney is restored to a more favorable situation through the adoption of a given position of the body or as the result of efforts at reposition. The occurrence of the change for the better is often manifested by the evacuation of large amounts of urine within a short time. At the same time the kidney becomes greatly reduced in size. It is noteworthy that in women the symptoms of wandering kidney are generally intensified at the **menstrual period.** I have had under observation women in whom wandering kidney could be demonstrated distinctly almost only at the menstrual period. The demonstration of a wandering kidney during life is therefore of importance, because the condition might be readily overlooked after death if the kidney has accidentally been restored to its usual situation when the body is placed in the recumbent posture.

Prognosis.—Wandering kidney is a troublesome and frequently an incurable condition, although, as a rule, it is not attended with danger to life. Symptoms of incarceration will only exceptionally be followed by death from uremia. In the prognosis the observation is worthy of consideration that a wandering kidney exhibits a tendency to carcinomatous degeneration.

Treatment.—As a prophylactic measure, the wearing of tight clothing and constricting waistbands and belts should be strictly forbidden. Not much can be accomplished with internal remedies in the treatment of movable kidney. Only in emaciated persons will **rest in bed** and a **fattening diet** sometimes be followed by replacement of a wandering kidney as soon as the fatty capsule of the organ has been sufficiently developed and gives the organ greater support. Naturally, this result can be expected only in cases that are not too far advanced. Individuals with wandering kidney should avoid bodily strain and secure an easy evacuation of the bowels daily. Women should not lace their clothing tightly, but should wear it suspended from bands passing over the shoulders. The wearing of an **abdominal bandage** may be recommended, surrounding the abdomen from the umbilicus downward, and by pressure forcing the kidney upward toward its natural position. Bandages with compresses, designed directly to force the kidney upward, rarely fit well. Often the movable organ slips from beneath the compress, and the bandage then does more harm than good. At times wandering kidney has been relieved by **massage.** In cases in which the symptoms are aggravated **surgical treatment** has been attempted. The most certain procedure consists in removal of the movable kidney—*nephrectomy;* but this will preferably not be undertaken, because it is not rarely observed that the function of the other kidney fails, and death results from uremia. As a rule, it will therefore suffice to suture ie kidney in its normal situation—*nephrorrhaphy.* Naturally, it may happen that after some time the kidney will again become movable, and permanent relief is not afforded. Such *complications* as may arise should be treated according to the usual rules. In cases of incarceration of a wandering kidney efforts should be made to replace the organ, hot cataplasms should be applied, warm tea should be drunk, and, if pain be considerable, morphin may be administered subcutaneously.

Dystopia of the kidney is the designation given to an immovable displacement of the kidney which may be either congenital or acquired. Congenital displacement of the kidney is generally left-sided. At times the organ is found in the true pelvis. By causing contraction of the pelvis, obstruction to labor may result. Acquired displacement of the kidney may occur in connection with wandering kidney if the organ becomes attached in an abnormal situation, but sometimes it occurs also as the result of pressure upon the kidney by adjacent organs.

HORSESHOE KIDNEY.

Horseshoe kidney results from the coalescence of the two kidneys into a single organ. Generally the two lower poles of the kidneys are united, so that a semilunar or horseshoe-shaped body results, with its concavity directed upward. The ureters traverse the anterior aspect of the body downward. The coalescence of the kidneys is associated with their displacement, for generally the two organs are situated upon the anterior aspect of the lumbar vertebræ. Occasionally, the kidneys coalesce at the margins of the hilus. In this way there results a disc-shaped or cake-shaped body, which may possess a single pelvis, but giving off two ureters. Symptoms are usually absent, and often the condition is discovered either accidentally on careful examination of the abdominal cavity, or possibly only upon post-mortem examination. The diagnosis is by no means easy, and other abdominal tumors must, in the first place, be excluded. Besides, it may be found that the loins are depressed, and on percussion yield a tympanitic note.

ABSENCE OF THE KIDNEY.

At times one kidney may be absent, and the knowledge of this fact is naturally of the greatest importance in considering the operation of nephrectomy. The condition may be congenital or acquired. Thus, the kidney has occasionally been found transformed into a mass of fat.

Supernumerary kidneys are without clinical significance.

INFLAMMATION OF THE PARARENAL CONNECTIVE TISSUE (PARANEPHRITIS).

Etiology.—Inflammation of the loose and partly fatty connective tissue surrounding the kidney scarcely occurs other than as a result of the activity of bacteria, and among the exciting agents thus far isolated are the Streptococcus pyogenes, Staphylococcus pyogenes, Bacterium coli, tubercle-bacilli, pneumonia-cocci, and actinomyces. **Exposure to cold** may be a *contributing factor* in the development of *infection of the pararenal connective tissue* by bacteria, although the occurrence of a refrigeratory (rheumatic) paranephritis can scarcely be accepted as demonstrated with certainty. On the other hand, **traumatic paranephritis** undoubtedly occurs. Not only gunshot-wounds, stab-wounds, and open wounds generally may be mentioned in this connection; but falls and blows upon and other injuries to the loin, and violent concussion of the body, and even heavy lifting, may be operative. At times paranephritis may follow antecedent **infectious disease**, as, for instance, typhoid fever and septicopyemia. Most commonly paranephritis arises by **extension from adjacent inflammation.** It often develops in the sequence of pyelonephritis (particularly with urinary calculi), carcinoma, tuberculosis, and echinococcus of the kidney, and at times of embolism of the kidney. Paratyphlitis also may extend upward, and excite paranephritis. At times ulcers in the ascending or descending colon rupture into the pararenal connective tissue, and give rise to paranephritis. In addition, parametritis, periproctitis, carcinoma and tuberculosis of the vertebræ, abscess of the liver and of the spleen, duodenitis, psoitis, and purulent pleuritis may be mentioned as causative conditions. *Men* suffer most frequently from paranephritis. The disorder is less common in childhood than in adult life.

Anatomic Alterations.—The principal alterations of paranephritis consist in purulent infiltration of the pararenal connective tissue, which, however, soon becomes transformed into a collection of pus. The latter generally does not become encapsulated, and often the pus is admixed with tissue-detritus. Not rarely it possesses putrid properties. The kidney is usually marked by great mobility. At times paranephritis is complicated by perinephritis, and inflammation, and even accumulation of pus, may take place within the capsule of the kidney. The inflammatory process may also extend to the tissue of the kidney itself, and cause purulent destruction of this organ. At times inflammation and erosion of the vertebræ and purulent infiltration and destruction of adjacent muscular structures have been observed.

Symptoms and Diagnosis.—The symptoms of paranephritis are in part local and in part general. Only rarely does paranephritis set in like an acute infectious disease, with a chill and high fever. Far more commonly the disease begins insidiously. Often, complaint is first made of **pain in the loin,** which is usually increased upon pressure. The patients generally assume a **constrained attitude,** walking with the upper portion of the body bent forward and toward the diseased side; or in bed they lie upon the diseased side, holding the vertebral column convex toward the healthy side, with the lower extremity upon the diseased side flexed at the hip-joint and the knee-joint. The diagnosis cannot be made with certainty until a **visible swelling** and **palpable resistance** have made their appearance **in the loin.** The increased resistance generally appears earliest, and becomes particularly distinct upon bimanual palpation of the kidney. At first it involves rather the lumbar region proper, but it subsequently extends gradually toward the side and forward. A distinct limitation of the area of resistance is generally wanting, and becomes apparent only after the inflammatory process has existed for a considerable period of time. The side upon which the diseased kidney is situated often bulges posteriorly. The skin is often edematous and reddened. Derangement of micturition is usually wanting, and the urine itself is generally unaltered. As a rule, irregular **fever** is present, and chills may occur from time to time.

A common complication is *pleurisy* (generally serous) upon the same side of the body. The *course of paranephritis* may extend over from four to six weeks, or even from two to four months, and still longer, and a distinction accordingly is made between acute, subacute, and chronic paranephritis. The disease may terminate in complete *recovery* by absorption of the products of the inflammatory process, but more commonly *rupture of the pus* takes place unless anticipated by surgical intervention. Rupture of the pus may take place into the stomach, the intestine, the pelvis of the kidney and the ureter, the pleura and the bronchi, the abdominal cavity, or externally. In the last-named event extensive burrowing often takes place. The attending dangers are of variable degree. Rupture into the abdominal cavity will generally give rise to rapidly fatal peritonitis. Long-continued suppuration is attended with danger of *amyloid disease* and *fatal exhaustion.*

Prognosis.—The prognosis of paranephritis is not unfavorable if the accumulation of pus be incised and evacuated at the proper time.

Treatment.—As long as fluctuation cannot be elicited over the area of inflammation an attempt should be made to restrain the progress of the inflammatory process by the application of an **ice-bag to the loin,** or to stimulate absorption of inflammatory products by the application of **hot cataplasms.** As soon as fluctuation can be elicited there is but one remedy, namely, **incision.** Further, it may be exceedingly difficult to appreciate deep-seated fluctuation, so that resort has been had to exploratory puncture.

ANEURYSM OF THE RENAL ARTERY.

Aneurysm of the renal artery is a rare condition. It is often unrecognized during life. The patient dies suddenly from *internal hemorrhage,* the source of which is discovered upon post-mortem examination to be a ruptured aneurysm of the renal artery. Only rarely will a *tumor with expansile pulsation* be palpable in the renal region. More commonly symptoms of *renal colic* and *hematuria* are present, so that the clinical picture is suggestive of renal calculus.

Treatment is ineffective.

II. DISEASES OF THE PELVIS OF THE KIDNEY AND OF THE URETER.

DILATATION OF THE PELVIS OF THE KIDNEY (HYDRONEPHROSIS).

Etiology.—Dilatation of the pelvis of the kidney is invariably the result of **urinary stasis.** In accordance with the seat of the causes of the stasis hydronephrosis may be unilateral or bilateral, the latter particularly if the causes are related to disease of the bladder or the urethra. A common cause for hydronephrosis is constituted by **renal calculi,** which have formed in the pelvis of the kidney and have found their way into and obstructed the ureter. A similar result may be brought about by **echinococci, carcinoma, and tuberculosis of the kidneys** and of the pelvis of the kidney, if echinococcus-vesicles, carcinomatous tissue, or cheesy masses have become detached and are impacted in the ureter. Also, in cases of **pyelitis and hemorrhage into the pelvis of the kidney,** blood-clots or plugs of pus not rarely cause obstruction of the ureter and give rise to hydronephrosis. Obstruction is but rarely due to **parasites.** Among the **diseases of the bladder,** paralysis and tumors particularly may be mentioned as causes of hydronephrosis. Of **diseases of the urethra,** all varieties of constriction and obstruction must be taken into consideration. An associated group of causes includes the **compression-stenoses of the urinary passages,** which not rarely give rise to hydronephrosis. Among the more common of these conditions peritoneal exudates, displacements and neoplasms of the uterus, ovarian tumors, carcinoma of the rectum, and prostatic hypertrophy may be mentioned. Hydronephrosis resulting from *torsion* or *kinking of the ureter* has already been considered in the discussion of the symptoms attending incarceration of a wandering kidney (p. 430). Hydronephrosis may further be of *congenital* origin. The causes of this character consist at times in abnormal valvular formations at the junction of the ureter with the pelvis of the kidney or with the bladder, and at other times in unusual length of the course of the ureter within the wall of the bladder. Also, congenital atresia of the ureter, the bladder, or the urethra should be mentioned in this connection.

Anatomic Alterations.—The most important anatomic alteration attending hydronephrosis is naturally the dilatation of the pelvis of the kidney, and which may attain such a marked degree as to equal or even exceed the size of an adult head. When the pelvis of the kidney becomes so greatly enlarged the related kidney itself must naturally be involved. The latter becomes compressed, flattened, and stretched, and at times consists only of a thin band of renal tissue. The ureter itself may also participate in the dilatation, and at times attains the size of a loop of small intestine, and, like this, it may form several convolutions.

At times, in the presence of abnormal valvular formations in the pelvis of the kidney, hydronephrosis may be confined to one or more renal calices —*partial hydronephrosis*—and it will be recognizable from the flattening and compression of the corresponding renal papillæ.

The contents of the dilated renal pelvis consist of stagnant urine, which is either unchanged or, in consequence of inflammation of the pelvis of the kidney, has acquired a purulent, at times a gelatinous, or, as a result of admixture of blood, a reddish, reddish-brown, almost chocolate-like appearance. If complete obstruction of the ureter has existed for a considerable length of time, urea and uric acid may disappear from the fluid. In some cases obstruction to the flow of urine can scarcely be demonstrated with certainty, as reflections and valvular formations readily become obliterated in the dead body, and may perhaps be apparent only under certain conditions of distention and in certain positions.

Symptoms.—It is useful to separate the symptoms of *congenital* from those of *acquired hydronephrosis. Congenital hydronephrosis* may at times attain such considerable proportions as to constitute an **obstruction to labor**, and which can be overcome only by puncture or even by embryotomy. If a child with congenital hydronephrosis is born alive, it will exhibit frequently dyspnea and cyanosis, because the diaphragm, the lungs, and the heart are displaced upward, and death may result within a short time in consequence of asphyxia or cardiac paralysis. Death may result also from uremia. Not rarely the little patient presents also other malformations, as, for instance, harelip, cleft palate, clubfoot. At times congenital hydronephrosis, which further is often bilateral, is observed in several children in the same family.

Acquired hydronephrosis is recognized during life only when a **tumor in the loin** can be demonstrated which while under observation gradually increases in size, while at the same time stasis-producing conditions are present in the urinary passages. Frequently, but by no means constantly, **fluctuation** can be elicited over the renal tumor. Of especial importance in the diagnosis is palpation of the dilated ureter, but this is rarely possible. At times the patients complain of **pain in the renal region,** and tenderness on pressure may be present in the same situation. The more rapidly hydronephrosis develops and the more extensive it becomes, the more intense is the renal pain likely to be. The changes in the **amount** and the **constitution of the urine** are noteworthy, for if large amounts of urine collect in the pelvis of the kidney within a short time, the amount of urine excreted will appreciably diminish. Naturally, the sudden disappearance of hydronephrosis will be revealed by rapid increase in the amount of urine. When the urine is rendered turbid by admixture with pus, it becomes clear when the ureter of the inflamed renal pelvis is obstructed, and the entrance of purulent urine into the bladder is prevented, with simultaneous development of hydronephrosis. Under such conditions, with the removal of the obstruction, disappearance of the hydronephrosis, and increase in the amount of urine, the urine again becomes turbid. Not rarely, in consequence of urinary stasis, **uremic** and **urinary septic symptoms** appear, particularly chills, febrile movement, nausea, vomiting, headache, even convulsive attacks. These symptoms are observed with particular frequency when hydronephrosis is bilateral, and then death may ensue from uremia.

The *duration of hydronephrosis* depends upon the causative factors in the individual case. At times the condition persists for a few hours or days—*acute hydronephrosis;* at other times for years, or throughout life—*chronic hydronephrosis.* In accordance with the course of the disease, several *varieties of hydronephrosis* have been distinguished. The designation *intermittent hydronephrosis* has been applied to cases in which the factors causative of urinary stasis are frequently repeated, and each time give rise to hydronephrosis, which disappears with the removal of the obstruction. The designation *remittent hydronephrosis* is applied to cases in which the condition persists continuously, and from time to time undergoes increase and then diminution. A reduction in the hydronephrosis takes place when the urine in the dilated renal pelvis becomes subjected to such a considerable degree of pressure that this is capable of partially overcoming the obstruction and permitting partial evacuation of the renal pelvis. Accordingly as the flow of urine from the pelvis of the kidney is completely or partially interfered with, a distinction is made between *open* and *closed hydronephrosis.* In addition to the dangers of uremia and septicemia already mentioned, there is, besides, a possibility of *rupture of the dilated renal pelvis,* but this occurrence is rare.

Diagnosis and Prognosis.—The recognition of hydronephrosis is not easy even when a *renal tumor* is demonstrable. In the diagnosis the demonstration of **conditions capable of causing**

urinary stasis is decisive. It is therefore necessary in every case to endeavor to determine these, because the prognosis and the treatment will be governed accordingly.

Treatment.—The treatment of hydronephrosis can be only **mechanical**. Efforts should be made to remove the causes of the urinary stasis. Should life be threatened in consequence of the extent of the hydronephrosis or of suppuration, **nephrotomy** or **nephrectomy** will have to be decided upon, in accordance with the condition of the related kidney. Further, nephrectomy is not rarely complicated by serious difficulties from the fact that the dilated renal pelvis is often intimately adherent to the stomach or to adjacent loops of intestine.

INFLAMMATION OF THE PELVIS OF THE KIDNEY (PYELITIS).

Etiology.—Pyelitis results from either **bacterial** or **toxic influences**, most commonly from the former. The exciting agents of the inflammatory process include the same bacteria that have been mentioned as the causes of suppurative nephritis, and this fact is readily explained from the circumstance that pyelitis and nephritis are frequently associated—so-called *pyelonephritis*. Accordingly, in addition to the ordinary pyogenic microörganisms (Streptococcus pyogenes, Staphylococcus pyogenes), the Bacterium coli particularly, and, further, also pneumoniacocci, tubercle-bacilli, and typhoid-bacilli must be mentioned. Frequently bacteria gain entrance from the bladder into the ureter and the pelvis of the kidney, and pyelitis thus readily becomes associated with *inflammatory disorders of the urinary bladder and the urethra*. The danger is particularly great when urinary stasis results. At other times pyelitis is the result of **disease of the renal pelvis** itself. It occurs with especial constancy in connection with renal calculi and with tuberculosis, carcinoma, and parasites of the renal pelvis. Little of a definite nature is known with regard to refrigeratory (rheumatic) pyelitis, while, on the contrary, traumatic pyelitis may undoubtedly occur after a fall, blow, or injury in the renal region. The traumatism, however, has only the significance of a contributory cause for the infection of the pelvis of the kidney with bacteria. Pyelitis is often associated with **diseases of the kidney**. It is a frequent phenomenon in the presence of hypostasis of the kidney, acute diffuse nephritis, and the remaining forms of diffuse nephritis, suppurative nephritis, carcinoma, tuberculosis, and echinococcus of the kidney, etc. At times pyelitis arises by **extension from adjacent disease**, and it may complicate paranephritis and peritonitis. In some cases pyelitis develops in the sequence of **infectious diseases**, as, for instance, small-pox, typhoid fever, pneumonia, erysipelas, scarlet fever, etc. *Toxic pyelitis* may develop after the ingestion and the employment of all those remedies that have been mentioned on pp. 402 and 403 as causes of acute nephritis, and among which particularly mineral acids, carbolic acid, balsamics, and cantharides may be mentioned here. Pyelitis is rare in children.

Anatomic Alterations.—Inflammation of the mucous membrane of the pelvis of the kidney is attended with **hyperemia, swelling, and increased secretion of the mucous membrane.** Here and there also small extravasations of blood have taken place into the mucous membrane. So long as the condition is one only of increased secretion of mucus and possibly excessive desquamation of the epithelium of the mucous membrane it can be designated a mucous or desquamative catarrh of the renal pelvis. In some cases, however, there is active formation of pus, so that the condition becomes one of purulent pyelitis. Considerable hemorrhage from the surface of the mucous membrane gives rise to the clinical picture of hemorrhagic pyelitis. Toxic causative factors may give rise to a fibrinous pyelitis. A necrotizing pyelitis develops when the mucous membrane of the renal pelvis throughout a greater or lesser extent is converted into a necrotic, friable grayish-green or blackish tissue. Secondary involvement of the kidneys in the inflammatory process is readily possible from the fact that bacteria may gain entrance to the uriniferous tubules directly from the renal pelvis, and from this point may excite inflammation. Generally the *ureters* also are involved in the inflammatory process. Pyelitis may be *unilateral* or *bilateral*, accordingly as the causative factors are operative upon one side or upon both, being unilateral, for instance, in the presence of renal calculi, parasites, and paranephritis.

Symptoms and Diagnosis.—A positive diagnosis of pyelitis is often impossible, and in the majority of cases more than a tentative diagnosis, upon an empirical basis, cannot be made. If pyelitis is associated with disease of the kidneys, bladder, or urethra, the urinary alterations of the former condition are often obscured by those of the renal, vesical, or urethral disorder. In cases of pyelitis of rather independent origin attention should be directed particularly to **local alterations in the kidneys** and to **changes in the urine.** Patients with pyelitis often complain of a sense **of tension,** of **pressure,** and even of **pain in the renal region,** which, when the pyelitis is unilateral, may be referred only to the corresponding side. Pressure in the renal region is likely to increase the pain, or may induce it. The *changes in the urine* vary with the nature of the inflammatory process. In the mildest cases the urine contains only an increased amount of mucus, so that the cloudiness of normal urine is increased in amount and density. On microscopic examination of the urinary sediment attention may be attracted at times by caudate or club-shaped epithelial cells, which may be derived from the deeper epithelial layers of the urinary bladder, but which are rather suggestive of pyelitis when large squamous epithelial cells are wanting in the urinary sediment, and when the cells present are abundant and arranged in layers like shingles on a roof. In cases of purulent pyelitis the urinary sediment usually contains polynuclear pus-corpuscles. As long as the kidney is not involved in the inflammatory process the filtered urine will contain no albumin, or only traces thereof. Also, no tube-casts are found in the sediment. Similar alterations in the urine attend cystitis and urethritis, but the pain in the renal region is often distinctive of pyelitis. In cases of hemorrhagic pyelitis *hematuria* occurs, and the urine may contain cylindric coagula that have formed in the ureter. Pyelitis is often associated with *hydronephrosis*, because the ureter is obstructed by collections of pus-corpuscles or blood-clots. Under such conditions severe renal pain occurs suddenly, with chill, fever, vomiting, and diminution in the amount of urine, which becomes clear; these symptoms soon subside when the urinary obstruction is relieved.

In accordance with the causative factors pyelitis may pursue an *acute*, a *subacute*, or a *chronic course*. Chronic pyelitis is generally purulent in character, is associated as a rule with purulent nephritis, and not rarely causes death by uremia, urinary septicemia, rupture into the neighborhood, or amyloid disease.

Prognosis.—The prognosis of pyelitis depends upon the curability of the causative conditions.

Treatment.—Every case of pyelitis requires, in the first place, **causal therapy.** Under all conditions the patient should be kept **in bed**, in order to maintain a uniform temperature, and be given **fluid to drink in abundance**, in order to dilute the urine as largely as possible and to avert urinary stasis in the ureter. For this purpose well-boiled milk, to which lime-water (1 tablespoonful to each glass of milk) is added, red wine diluted half with water, and tea, particularly an infusion of uva ursi leaves (1 tablespoonful to a large cupful of hot water three or four times daily), may be particularly recommended. If thirst be marked, alkaline waters may be prescribed (Wildungen, Fachingen, Ems, Vichy), or carbonated waters (Bilin, Giesshübel). For the relief of pain in the renal region **hot cataplasms** may be applied. Internally **disinfectants**, **balsamics**, or **astringents** may be administered. Personally I usually begin with salol, and after from ten to fourteen days substitute oil of turpentine, and finally prescribe tannic acid:

℞ Salol,	1.0 (15 grains);
Saccharin,	0.02 (⅓ grain).—M.

Make 10 such powders.
Dose: 1 powder every two hours.

℞ Oil of turpentine, 10.0 (2½ fluidrams).
Dose: 10 drops in milk thrice daily.

℞ Tannic acid, 0.3 (4½ grains);
Saccharin, 0.02 (⅓ grain).—M.
Make 10 such starch-capsules.
Dose: 1 capsule every three hours.

Profound involvement of the kidneys in the inflammatory process may require nephrotomy or nephrectomy, as has been mentioned on p. 420.

RENAL CALCULI (NEPHROLITHIASIS).

Anatomic Alterations.—The pelvis of the kidney is a frequent seat for the deposition of earthy precipitates from the urine. In accordance with their size a distinction has been made between *renal sand* and *renal calculi*, the latter designation being applied only to grains of precipitate larger than a pin-head in size. *Chemically*, calculi may consist of urates, phosphates, oxalates, carbonates, xanthin, cystin, or indigo.

In some instances *fibrinous concretions* have been encountered in the pelvis of the kidney. These have resulted from antecedent hemorrhage, and consist of tough, flexible, and extensile structures that are inflammable and yield the odor of burning feathers.

Uratic calculi consist of uric acid, or generally of its salts (urates). They are the most common variety of renal calculi, are generally hard, and sometimes possess a smooth and at other times a granular surface. In color they vary from brown to yellow. Upon transverse section dark and light layers often alternate with each other. Occasionally the nucleus consists of calcium oxalate. The cortex at times is formed of earthy phosphates. Uric acid and urates can be readily recognized by means of the *murexid-test*.

Phosphatic calculi consist of calcium phosphate and ammonio-magnesium phosphate (triple phosphate), and but rarely of the latter alone. That phosphatic deposits are occasionally present in the cortex of uratic calculi has already been mentioned. Phosphatic calculi are friable, at times smooth, at other times nodular, and they vary in color between gray, yellowish red, and reddish violet.

Oxalate-calculi consist of calcium oxalate, and are generally characterized by a multinodular surface, so that they have also been designated *mulberry-calculi*. In color they are frequently deep black, because their sharp prominences give rise to considerable hemorrhage. Their degree of hardness is considerable.

Carbonate-calculi are but rarely found in human beings. They possess a whitish or a reddish color, and are readily recognized from the fact that the calcium carbonate is dissolved in hydrochloric acid with effervescence and the generation of carbon dioxid.

Xanthin-calculi likewise are rare. They vary in color from yellowish brown to dark brown, and the fractured surface, when rubbed, yields a waxy luster.

Cystin-calculi are hereditary in some families, and are among the less common varieties. They vary in color between white, yellow, and dark gray, although they may be green or blue. Their surface is at times smooth, at other times rough. Occasionally the nucleus consists of uric acid, and the cortex of earthy phosphates. Scrapings from the calculus are soluble in potassium hydroxid and ammonia, and on evaporation six-sided plates of cystin crystallize out.

Indigo-calculi occur but rarely. They are conspicuous for their bluish-black color, and for staining paper over which they are drawn.

The *size*, the *number*, and the *shape of renal calculi* exhibit remarkable variations. In general the size of renal calculi will be the greater the smaller their number and the greater room for growth they have. Renal calculi as large as a hen's egg have been observed. In general, the roundish or cylindric shape preponderates. At times a single calculus almost wholly occupies the entire pelvis of the kidney, and some of the processes may extend into the calices of the kidney, so that shapes result that have been appropriately compared with the appearance of the antlers of a deer. At times renal calculi develop within the calyx of a kidney, and in consequence acquire a shape suggestive of the appearance of a fumigating pastil. In some cases the pelvis of the kidney represents a sac filled with numerous small calculi, which may reach into hundreds. In association with renal calculi there is generally **inflammation of the renal pelvis**, which it is customary to designate as *calculous pyelitis*. As a rule, the inflammation is chronic in character, and generally purulent in nature. At times necrosis and ulceration of the mucous membrane of the renal pelvis occur. The ulcers may perforate into adjacent organs, into the abdominal cavity, or externally, and give rise to the formation of a *renal-pelvic fistula*. Renal calculi are, further, not without influence upon the *kidney* itself. At times contracted kidney may develop, while in other instances purulent inflammation of the pelvis of the kidney may extend to the renal tissue and give rise to purulent nephritis. Decomposition of the urine within the pelvis of the kidney may readily cause involvement of the *ureter* and the *bladder*, with the development of chronic inflammation. In addition, these structures may suffer when a renal calculus is expelled from the pelvis of the kidney and attempts to make its way downward.

Etiology.—Renal calculi are particularly common in *childhood* and in *advanced life*. They have even been observed in some instances in the newborn. Experience has shown that renal calculi are much more common in *males* than in females. A **sedentary mode of life** favors the formation of renal calculi in some inexplicable manner. The condition is almost unknown in sailors and soldiers. Renal calculi occur much more commonly in some regions than in others. England and Holland are well known as favorable soils for the formation of renal calculi. It is a remarkable fact that regions in which renal calculi are common may adjoin without sharp limitation regions in which renal calculi do not occur. In what the **geographic influence** consists is as yet wholly unknown. Without doubt, the **food** has an influence upon the formation of renal calculi, as persons are known who invariably present signs of renal calculi within a short time after taking a small amount of white wine. Not without reason the excessive use of meat and alcohol is held to favor the development of renal calculi. At times the formation of renal calculi is dependent upon **disorders of metabolism.** Gouty patients are not rarely the victims of renal calculi. Individuals suffering from cystinuria not rarely have cystin-calculi. Cystinuria and cystin-calculi are hereditary in some families, and thus it will be seen that an inheritance of anomalies in metabolism may give rise to an **inheritance of renal calculi.** At times renal calculi form about **foreign bodies** (blood-clot, ova of parasites). They may therefore follow **injuries** in the renal region if hemorrhage into the renal pelvis has taken place. The formation of renal calculi is favored in marked degree by all inflammatory diseases of the urinary passages, particularly if these are attended with urinary stasis and decomposition.

Little is definitely known with regard to the intimate *processes concerned in the formation of renal calculi.* The precipitation of concretions from the urine is theoretically possible under two conditions, namely, either if the urine contains so much of any material that it cannot retain all of it in solution, or if the reaction of the urine becomes so altered that certain substances are thrown out of solution. The simple precipitation from the urine of substances capable of forming calculi by no means, however, alone gives rise to the formation of renal calculi, for microscopic, and particularly microchemic, examination of renal calculi has shown that the calculus-forming substances have not simply crystallized together, but that they are bound together by an organic framework. This latter probably is scarcely other than the product of a catarrhal state of the mucous membrane of the renal pelvis, so that some clinicians have referred directly to a calculus-generating catarrh. As, however, bacteria may play a causative *rôle* in the development of such a catarrh, one is forced to the conclusion that, as in the development of gall-stones, so also in the formation of renal calculi, *bacteria* are of great importance. In the case of calculus-formation in decomposed urine bacteria again are concerned, and they must be looked upon as the cause of the alkaline decomposition. In this way is explained the great influence that all conditions of urinary stasis exert upon the formation of renal calculi, for whenever urinary stasis exists excessive development of bacteria in the urine is possible.

Symptoms and Diagnosis.—Renal calculi and renal sand may be present in the pelvis of the kidney without giving rise to any symptom, and they may be found quite accidentally upon post-mortem examination—*latent renal calculi.* In other instances symptoms of **pyelitis or pyelonephritis** appear, and that these are dependent upon renal calculi may be first discovered upon operative intervention or after rupture externally with the discharge of renal calculi. The circumstance would naturally be suspicious if the urine contained considerable crystalline sediment, and from the character of this it might be possible to judge of the chemic constitution of the calculus. Not rarely **pain in the renal region** is present, and this may radiate for quite a distance, and not at all rarely it is for a long time mistaken for lumbago, intercostal neuralgia, or the like. Renal calculi at times give rise also to a peculiar constrained **posture of the body.** The patients walk with the body bent forward, with the shoulder depressed toward the affected side, and the vertebral column convex toward the healthy side, avoiding from fear all rotatory and flexor movements of the vertebral column, and they walk as stiffly as a stick, placing the feet with care gently upon the floor, with especial concern for the foot upon the diseased side. At times the patients complain for weeks and months of **nausea** and **vomiting** before it becomes certain that these symptoms are due to reflex irritation by calculi in the pelvis of the kidney. The presence of renal calculi may be concealed also behind the clinical picture of *chronic catarrh of the urinary bladder.*

Some patients suffer repeatedly from **hematuria,** as the cause of which renal calculi may be suggested from the presence of considerable crystalline sediment in the urine. One of the most common morbid manifestations is **renal colic.** This occurs, however, only when calculi leave the pelvis of the kidney in an effort to pass through the ureter into the bladder, and remain impacted in the ureter. The patients then complain, in general suddenly, of intense pain in one renal region, setting in either without appreciable cause or induced by bodily over-exertion or concussion, emotional disturbance, indiscretion in eating and drinking, or the like. The pain not rarely radiates to remote parts, particularly toward the bladder, and even to the glans penis and the thigh. The character of the pain is variously described, and is almost uniformly designated by all patients as overwhelming. The facial expression is, therefore, one of fear and pain, and the skin in consequence of reflex vascular spasm is pale and cool, and frequently covered with cold and clammy sweat. If the cerebral vessels are involved in the spasm of the vascular muscles, syncope and clonic muscular spasm may result. Often chill, fever, and vomiting occur.

Particularly noteworthy further are **changes in the urine** at the time of the attack of colic. As a rule, the urine is passed in diminished amount, and in some cases **anuria occurs,** although but one ureter is occluded, because the function of the healthy kidney is suppressed in consequence of reflex irritation. Should the anuria persist for several days, danger of uremia may arise, with the possibility of a fatal termination. Frequently the urine is bloody—*hematuria;* this occasionally persists for a considerable time, and may be quite abundant. It is due to injury of the mucous membrane of the renal pelvis by the impacted calculus. If the lumen of the ureter again becomes free, from either the return of the calculus into the pelvis or its entrance into the bladder, the renal pain ceases, the urine again becomes abundant, and gradually the blood disappears. Naturally, severe pain in the bladder and urethra may be renewed when the calculus begins its passage from the bladder through the urethra on its way to the outside.

The *duration of an attack of renal colic* is subject to great variation, and may be from a few hours to several days. As a rule, renewed attacks of renal colic occur after a longer or shorter interval, because frequently several calculi are present in the pelvis of the kidney, or the calculi have returned from the ureter into the pelvis of the kidney, subsequently to resume their passage to the ureter. Not rarely patients suffer from attacks of renal colic for many years and, at times with greater, at other times with less severity, even throughout life. An attack of renal colic is not, as a rule, fatal. Serious dangers may arise when a renal calculus cannot pass upward or downward in the ureter, for there may then result inflammation, gangrene, and rupture of the ureter, with rapidly fatal peritonitis. If a renal calculus completely occludes the ureter, the urine stagnates in the pelvis of the kidney, and *acute hydronephrosis* develops, and at times gives rise to a palpable renal tumor.

That an attack of renal colic is due to the presence of a calculus will become demonstrated with certainty only when renal calculi are passed with the urine. Suspicion will, naturally, always be aroused if the urine contains considerable crystalline sediment, from whose nature conclusions may at the same time be formed as to the chemic constitution of the renal calculi. In rare instances calculi are passed with the urine without giving rise to any symptoms, most commonly in elderly women, but I have observed this occurrence at times also in men, although the calculi were almost as large as lentils. For this to take place the urinary passages must be of a certain size and the musculature be relaxed.

Prognosis.—The prognosis of renal calculi is not unfavorable in the sense that death but rarely results. With regard to permanent recovery, the conditions are naturally different, and frequently there is renewed formation of calculi as soon as the old calculi have been expelled. The conditions become serious when the kidneys are profoundly involved in consequence of the formation of calculi, in the form of either purulent pyelonephritis or contracted kidney.

Treatment.—The treatment of renal calculi varies in accordance with the chemic constitution of the precipitates. In the presence of *uratic calculi* experience has taught the wisdom of interdicting the use of alcohol as largely as possible, encouraging active physical exercise in the open air, and restricting the use of meat, although the earlier view is incorrect that generous use of meat gives rise directly to increased formation of uric acid in the body. All substances rich in nuclein-bodies (thymus, spleen, brain, lymphatic glands, liver, kidneys) are to be avoided particularly, because uric acid is derived from them. In order to diminish the acidity of the urine vegetables and fruit should be taken in abundance. It has of late become the fashion to eat oranges in considerable amount. It is well for the patient to drink daily a bottle of an alkaline or carbonated water, among which those of Fachingen, Obersalzbrunn, Ems, Vichy, Wiesbaden, Giesshübel, Bilin, may be mentioned. The use of tepid baths (28° R.—35° C.—95° F.) twice daily generally mitigates such symptoms as may be present. Attempts have further been made to effect **solution of uratic calculi** by medicaments, although the possibility of such a result has not been demonstrated with certainty, because these solvents pass into the urine in such small amount that a solution of excreted uric acid can scarcely be conceived. Among such solvents may be mentioned the following: lithium carbonate, lithium benzoate, lithium salicylate, lithium citrate, lithium bromid (of each, 0.5—7½ grains—thrice daily in a wine-glassful of soda-water), benzoic acid (0.5—7½ grains—thrice daily), magnesium borocitrate (1.0 : 100—15 grains : 3 fluidounces; 1 tablespoonful every hour), piperazin (0.5—7½ grains—thrice daily), lysidin (from 1.0 to 5.0 : 500—from 15 to 75 grains : 1 pint—of soda-water daily), uricedin (0.5—7½ grains—four times daily), urotropin (1.0—15 grains—thrice daily).

Well-to-do patients may be recommended courses of treatment at the **baths** during the summer, such as the alkaline baths of Carlsbad, Marienbad, Tarasp, Ems, Wiesbaden, Vichy, the lithium-springs of Assmannshausen, Baden, Salzschlirf, and the springs of Wildungen and Contrexeville. Individuals with *oxalate-calculi* should observe the same mode of life as those with uratic calculi, and they should be especially careful to avoid such vegetables as contain an abundance of calcium oxalate. These include particularly spinach, tomatoes, sorrel, rhubarb, cocoa, and tea. *Xanthin-calculi*, *cystin-calculi*, and *indigo-calculi* require similar therapeutic measures. The conditions, however, are quite different in the treatment of *phosphatic calculi* and *carbonate-calculi*. As these occur only in alkaline urine, efforts should be made to render the reaction of the urine acid. The ingestion of vegetables and fruit should therefore be restricted. In addition, mineral acids should be administered internally, as, for instance, hydrochloric acid and nitric acid:

℞ Dilute hydrochloric acid,	
Dilute nitric acid,	each, 2.0 (30 minims);
Distilled water,	180.0 (6 fluidounces);
Sirup of red raspberry,	15.0 (½ fluidounce).—M.

Dose: 15 c.c. (1 tablespoonful) every two hours.

A coexisting pyelitis naturally will also be favorably influenced by the employment of alkaline waters. If *renal colic* develops, a **hot cataplasm** should be applied to the loin, weak tea or an acid water should be taken abundantly in order to increase the elimination of urine and to favor the passage of the calculus, and, if the pain be severe, a subcutaneous injection of morphin may be given:

℞ Morphin hydrochlorate,	0.3 (4½ grains);
Glycerin,	
Distilled water,	each, 5.0 (75 minims).—M.

Dose: From 0.25 to 0.5 (4 to 8 minims) subcutaneously.

Of late, the **operative removal of renal calculi** has been undertaken with brilliant results. This matter will arise for consideration when the symptoms due to the calculus are so persistent and so severe as to render life unendurable to the patient. Under such circumstances the kidney and its pelvis will be incised —*nephrolithotomy;* or the pelvis alone will be opened—*pyelolithotomy*—and the calculi removed. In addition, impaction of a calculus in the ureter may furnish the indication for exposure of the ureter, with an effort to force the calculus upward or downward by massage with the fingers, or, should this fail, the ureter may be opened in order to remove the stone—*ureterolithotomy*. Finally, operation will be resorted to when the calculi in the renal pelvis have induced advanced pyelonephritis. In accordance with the condition of the kidney the operation of *nephrotomy* or of *nephrectomy* will then be performed. In cases of doubtful diagnosis *exploratory incision* of the kidney has even been practised.

CARCINOMA OF THE PELVIS OF THE KIDNEY AND THE URETER.

Primary carcinoma of the pelvis of the kidney or the ureter is uncommon. Generally the new-growth is a secondary carcinoma which has invaded the pelvis of the kidney from the kidney itself or from the neighborhood. The symptoms are those of carcinoma of the kidney.

PARASITES OF THE PELVIS OF THE KIDNEY.

Rarely the **palisade-worm—Strongylus gigas**—is found in the pelvis of the kidney. It resembles a spool-worm, except that it is supplied with six instead of three nipple-like papillæ around the buccal orifice. It gives rise to the symptoms of pyelitis, and causes pain in the kidney, vesical tenesmus, pyuria, and hematuria. In addition, the **Distoma hæmatobium**[1] also occurs in the pelvis of the kidney. It gives rise to the same clinical symptoms as the palisade-worm, and may be recognized from the appearance of distoma-ova in the urinary sediment. The eggs are oval, and in the neighborhood of one pole possess a lateral process. The parasites may be acquired by the ingestion in the Orient—as, for instance, in Egypt—of water containing ova.

III. DISEASES OF THE URINARY BLADDER.

INFLAMMATION OF THE URINARY BLADDER (UROCYSTITIS).

Etiology.—Inflammation of the urinary bladder is most commonly induced by **bacteria,** while **toxic urocystitis** is far less common. Among the bacteria Streptococcus and Staphylococcus pyogenes—particularly, however, the Bacterium coli, at times also tubercle-bacilli, gonococci, and proteus—may be mentioned as the exciting agents of the inflammatory process. Not rarely *mixed infection* has taken place, several varieties of bacteria occurring together. Among the **contributory causes for infection of the urinary bladder** with bacteria, exposure to cold should be mentioned in the first place—*refrigeratory* (*rheumatic*) *urocystitis.* Exposure of the abdomen while the body is the seat of perspiration, wetting of the clothing, cold baths and spongings, and insufficient clothing are not rarely assigned by the patient as causes of the disorder. **Traumatic urocystitis** may be induced by external or internal injuries; as, for instance, by a fall, a blow, a kick in the hypogastrium, the introduction of a catheter and other surgical instruments, even when these have been sterilized. Urocystitis arises with great frequency by **extension from adjacent disease.** Chronic gonorrhea involving the posterior portion of the urethra often causes urocystitis, and many cases of apparently spontaneous origin are dependent upon an insidious, almost forgotten chronic gonorrheal urethritis. Urocystitis, however, also frequently complicates acute gonorrheal urethritis, parametritis, and perimetritis, oöphoritis, proctitis, prostatitis, pyelitis, and nephritis.

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[1] Schistosoma hæmatobium.—A. A. E.

Previous disease of the bladder also is a common cause for urocystitis, which thus is encountered in association with vesical calculi and tumors and tuberculosis of the bladder. Urocystitis occurs with particular frequency in association with paralysis of the bladder, for when the sphincter vesicæ is paralyzed the possibility arises for the entrance of bacteria from the urethra into the bladder, with the development of inflammation. Urocystitis occurs at times in the course of **infectious diseases** (typhoid fever, pneumonia, articular rheumatism, cholera, dysentery, septicopyemia). Immediate infection of the bladder with bacteria may be effected by means of **catheters** and other **surgical instruments** if these are not freed from bacteria and sterilized before introduction into the bladder; but even the introduction of sterile instruments may not be unattended with danger if the urethra contains bacteria in considerable number, as these may be forced into the bladder by the instrument used. *Toxic urocystitis* is occasionally observed after the ingestion of cantharides, balsamics, or mineral acids. Also, the injection of irritating substances into the urethra not rarely gives rise to inflammation of the bladder. All conditions attended with *urinary stasis* favor in marked degree the development of urocystitis, because bacteria readily undergo multiplication in stagnant urine. For this reason the disorder is frequently observed in the train of stricture of the urethra and prostatic hypertrophy, during pregnancy, in association with flexion of the uterus, constipation, and after suppression of urine. Urocystitis is an uncommon disease in childhood, but it is particularly frequent in the aged. In men it is often dependent upon senile hypertrophy of the prostate gland. Experience has shown that men suffer from urocystitis more commonly than women.

Anatomic Alterations.—The mucous membrane of the urinary bladder when inflamed exhibits redness, swelling, and increased secretion of mucus. In the presence of acute urocystitis the color of the mucous membrane is bright red, while in the presence of chronic inflammation it is rather brownish red or reddish gray. Frequently, extravasations of blood have taken place into the mucous membrane, which then presents a bloody or brownish, mottled appearance, and in case of chronic inflammation a grayish or a blackish, mottled appearance. The swelling of the mucous membrane, in cases of acute inflammation, depends upon serous infiltration, while in cases of chronic inflammation it is due to inflammatory hyperplasia of the tissues of the mucous membrane. The inflammatory hyperplasia of the connective tissue not rarely extends to the muscular coat of the bladder, in consequence of which the latter becomes thickened and often projects into the cavity of the bladder in the form of numerous columns (trabecular degeneration), so that the interior of the bladder suggests the appearance of one of the ventricles of the heart.

Often the cavity of the bladder is altered, the viscus being either unusually capacious or remarkably small, and accordingly a distinction has been made between *eccentric* and *concentric hypertrophy of the bladder*. Occasionally some of the spaces between projecting columns of muscle have undergone bulging, and pockets (diverticula) of greater or lesser size have developed, which at times are larger than the cavity of the bladder itself, and occasionally contain urinary calculi. At times the serous membrane of the bladder also is involved in the inflammatory thickening, and presents a tendinous and turbid appearance.

Increased secretion of mucus in cases of inflammation of the bladder is attended with an abundant deposition of mucus and considerable admixture of mucus with the urine. The urine contained within the bladder usually presents a turbid, a mucous, a purulent, often also a bloody, appearance, and is frequently offensive in odor from alkaline decomposition. Severe inflammation of the bladder is at times attended with circumscribed accumulation of pus in the wall of the bladder—*abscess of the urinary bladder;* or there may be purulent inflammation of the serous coat of the bladder—*pericystitis*. That variety of urocystitis in which fibrinous membranes form upon the surface of the mucous membrane is upon the whole rare—*pseudomembranous urocystitis* (*fibrinous* or *croupous urocystitis*). Necrosis also occurs occasionally upon the mucous membrane—*necrotic* or *pseudodiphtheric urocystitis;* and this appears in the form of grayish-green or blackish masses, after exfoliation of which **ulcers** remain upon the mucous membrane. At times the ulceration extends deeply, and **rupture of the bladder** may take place, with the development of a **vesical fistula.** Rupture may take place into the vagina, the rectum, the peritoneal cavity, or externally.

Symptoms and Diagnosis.—In accordance with the duration of inflammation of the bladder a distinction is made between *acute* and *chronic urocystitis*. The latter may persist at times for many years. Both varieties naturally have in common the two most important symptoms—**derangement of micturition and alterations in the urine.**

Acute urocystitis is often first indicated by *difficulty in micturition*. The patients suffer from excessively frequent *vesical tenesmus*, and the act of micturition itself is attended with severe pain, with especial frequency and intensity at the conclusion of the act. If micturition is repeated at short intervals, only a few cubic centimeters of urine are voided each time, and frequently only a few drops. In addition, the patients often complain of a heavy and generally also markedly *painful feeling in the hypogastrium*, which is greatly increased by pressure in this region. At times *retention of urine* occurs, either because the mucous membrane in the neighborhood of the urethral orifice is greatly swollen as a result of the inflammatory process, or in consequence of spasm of the sphincter of the bladder. The *urine* presents a variable appearance in accordance with the nature of the inflammatory process. In the mildest cases, which may be designated *mucous urocystitis* (or less appropriately catarrh of the urinary bladder), the urine contains an excessive amount of mucus, so that, on standing, an exceedingly dense and extensive cloud of mucus forms at the bottom of the vessel. On microscopic examination of the urinary sediment a small number of round cells are found, and often a large number of desquamated epithelial cells from the mucous membrane of the bladder, so that the condition might be designated *desquamative catarrh of the urinary bladder*. At the same time the urine retains its acid reaction, and in some cases it may be even more than ordinarily acid.

The designation *suppurative urocystitis* (*purulent catarrh of the urinary bladder*) may be applied to cases in which the urine contains an abundance of pus-corpuscles—*pyuria*. On standing, a greenish-gray sediment collects at the bottom of the vessel, consisting, in addition to pus-corpuscles, of desquamated epithelial cells from the mucous membrane of the bladder. In the presence of purulent urocystitis also the urine often retains its acid reaction, while in other instances alkaline fermentation takes place, as indicated by the disagreeable, pungent, urinous odor. Under such conditions definite crystalline formations also frequently appear in the urinary sediment, especially the coffin-lid crystals of ammonio-magnesium phosphate (also known as triple phosphate), and the thorn-apple varieties of acid ammonium urate.

At times the pus is transformed into a viscid, mucoid mass, and on microscopic examination it is found that the pus-corpuscles are greatly swollen and transformed into almost homogeneous, polynuclear cells. Sometimes such a mass interferes with micturition, and if a catheter be introduced into the bladder the mass may be evacuated, often with great difficulty, when a greater or lesser amount of urine readily escapes. At times the urine acquires a *fecal odor*, obviously from the penetration into the bladder of gas from adjacent loops of bowel. The odor of hydrogen sulphid—*hydrothionuria*—has also been observed. Should the urine contain sugar, gas may be generated in the bladder from its fermentation—*pneumaturia*.

At times the urine contains *blood* in addition to pus—*hemorrhagic urocystitis*. In part the red blood-corpuscles are rather uniformly admixed with the urine, and impart to it a dirty-red, almost clay-like color, and in part blood-clots are found in the urinary sediment. Fortunately, the cases are rare in which necrotic vesical mucous membrane becomes detached and is evacuated with the urine in larger or smaller shreds. Pseudomembranous cystitis is at times attended with the presence of fibrinous membrane in the urine. The *amount of urine* in cases of inflammation of the bladder is often increased, probably in consequence of reflex irritation of the secretory nerves of the kidneys, and accordingly the specific gravity of the urine is diminished. The *general condition* may remain unchanged, apart from the fact that sleep may be greatly disturbed by the annoying tenesmus. In some cases, however, slight *fever* may be present. The patients lose their appetite, complain of increased thirst, and rapidly become pale and miserable.

Acute urocystitis may extend over a few days, or it may persist for from four to eight weeks. As a rule, recovery takes place gradually, the difficulty in micturition growing progressively less, while the urine becomes more and more clear, and regains its natural appearance. Should an abscess of the wall of the bladder develop, symptoms of severe general *septicemia* frequently appear, and should the urocystitis become complicated by a *peri-urocystitis* the region of the bladder will be extremely sensitive to touch.

Chronic urocystitis either develops in the sequence of acute inflammation of the bladder, if this recurs frequently, and particularly if recurrence takes place before recovery from the preceding attack has completely resulted; or it appears from the outset in a chronic and torpid form. The latter circumstance depends in part upon the causative factors. Thus, tumors, calculi, and tuberculosis of the bladder, as well as prostatic hypertrophy and stricture of the urethra, usually give rise to chronic urocystitis from the outset. Patients with chronic inflammation of the bladder complain, as a rule, of *vesical tenesmus* and *pain in micturition,* although the symptoms are generally less pronounced than in cases of acute urocystitis. The urine contains pus or blood, and gradually acquires an alkaline reaction. Fever is generally absent, except when acute exacerbations occur, while strength is gradually lost, and eventually death may result from exhaustion. At times putrid urinary products are absorbed from the bladder, and *urinary septicemia* develops (fever, stupor, sweats, exhaustion). The inflammatory process may also extend from the bladder to the pelvis of the kidneys, and give rise to *pyelonephritis* and its consequences.

Chronic urocystitis becomes particularly troublesome when complicated by *paralysis of the sphincter of the bladder.* The patients are then unable to retain the urine, which is passed into the clothing and the body-linen, and they diffuse a penetrating, urinous odor, while at the same time the soiled clothing acquires a reddish-yellow color where it is saturated, and in this way the condition of incontinence may become manifest even to the ordinary observer. When *eccentric hypertrophy of the bladder* has developed in the course of chronic urocystitis the bladder may be felt above the pubes and is never completely emptied. In the presence of *concentric hypertrophy of the bladder* the viscus must be emptied at short intervals, as it is capable of containing only a small amount of fluid, and, besides, the bladder can frequently be felt through the vagina or the rectum as a hard, globular body. The diagnosis of *acute and chronic urocystitis* is not difficult on account of the readily appreciable symptoms, but diagnostic difficulty may arise in the determination of the causes of the inflammatory process.

Prognosis.—The prognosis of inflammation of the bladder depends essentially upon the curability or incurability of the causative factors. Cryptogenetic chronic urocystitis in young persons deserves particular consideration, for it is often dependent upon tuberculosis.

Treatment.—The following measures for the **prophylaxis** of inflammation of the bladder are particularly to be mentioned: Avoidance of exposure of the abdomen when the body is heated, introduction into the bladder of only thoroughly sterilized instruments, careful and thorough treatment of urethritis, and care in the use of cantharides, balsamics, and other agents capable of causing irritation of the bladder. In the treatment of all varieties of urocystitis the first place should be given to the **mode of life** and the **diet**. Persons with acute catarrh of the bladder should remain in *bed*, and those with chronic catarrh should protect the hypogastrium against cold by the application of a woollen *abdominal bandage*. Sudden cooling of the hypogastric region is always injurious. The use of *baths*, at a temperature of 30° R. (37.5° C. —99.5° F.), for from fifteen to thirty minutes, from twice to four times weekly, may be advised. After the bath warmed clothing should be put on. The bed, also, should be warmed. All irritating *articles of food and drink* should be avoided, particularly strong alcoholics, beer, spices, acids, fruit, and asparagus, the last of which readily causes irritation of the urinary organs. The use of boiled *milk* is particularly to be recommended, to each glass of which a tablespoonful of *lime-water* may be advantageously added. The use of weak *tea* also may be recommended. If thirst be marked, a bottle of Vichy, Fachingen, Ems, Wildungen, Giesshübler, or Bilin water daily should be permitted. In cases of *acute inflammation of the bladder* internal treatment must suffice, and use be made particularly of **astringents, disinfectants,** or **balsamics.** Among *astringents*, I prefer a tea of uva ursi leaves, making use less commonly of tannic acid, arbutin, and other astringents:

℞ Uva ursi leaves, 100.0 (3 fluidounces).

Dose: 1 tablespoonful to a large cup of water as an infusion thrice daily.

In addition, *disinfectants* may be administered at the same time, and of these I prefer salol:

℞ Salol,	1.0 (15 grains);
Saccharin,	0.02 (⅓ grain).—M.

Make 10 such powders.
Dose: 1 powder every three hours.

Saccharin is advantageously selected as a corrective for salol, because antifermentative properties have been ascribed to it also. Should the urine acquire a greenish or blackish color after the use of salol, this will be no reason for the withdrawal of the drug; but the conditions are otherwise if albumin in considerable amount appears in the urine. The remedy may be continued for from ten days to two weeks. In my experience, salicylic acid (1.0—15 grains—every two hours) and sodium salicylate (1.0—15 grains—every two hours) are less reliable in action than salol.

Among *balsamics*, the first place should be given to oil of turpentine, which likewise may be employed for one or two weeks:

℞ Oil of turpentine, 10.0 (2½ fluidrams).
Dose: 10 drops in milk thrice daily.

When the urine is highly alkaline, *potassium chlorate* has been recommended, although my personal experience with it is by no means favorable:

℞ Solution of potassium chlorate, 10.0 : 200 (2½ drams : 6½ fluidounces).
Dose: 15 c.c. (1 tablespoonful) thrice daily after meals.

Potassium chlorate should be given only when the stomach contains food, and never when this viscus is empty, as in the latter event toxic symptoms might be readily induced (dissolution of red corpuscles and its consequences).

The internal treatment of *chronic inflammation of the bladder* is the same as that of acute cystitis, although in obstinate cases local treatment of the bladder with irrigations should be recommended additionally. Naturally, the latter will effect complete and permanent success only if previously the *causal indications* have been met, and, for instance, vesical calculi removed, prostatic hypertrophy or stricture of the urethra relieved, and the like.

For *irrigation of the bladder* an ordinary irrigator and a simple catheter are employed. Fluid at the temperature of the body and under moderate pressure is permitted to flow into the bladder until the patient complains of a sense of fulness or the fundus of the bladder can be felt above the symphysis of the pubes. The employment of a double-current catheter appears less serviceable, because the bladder is not distended and completely filled, so that some portions of the wall of the bladder and diverticula readily remain unirrigated. As the bladder when the seat of chronic inflammation often has a tendency to contract gradually and undergo progressive diminution in size, it is advantageous from time to time to cause marked distention of the viscus by completely filling it. As an *irrigating fluid* a solution of *silver nitrate* should be given first place. A weak solution (0.2 : 500—3 grains; 16 fluidounces) is begun with, and the strength is gradually increased to 5.0 : 500 (7½ grains : 16 fluidounces). *Tannic acid* and *boric acid* (from 2.0 to 10.0 : 500—from 30 grains to 2½ drams : 16 fluidounces) are also worthy of recommendation. If the urine contain large amounts of mucus, the bladder should be irrigated with *sodium chlorid* (5.0 : 500—75 grains : 16 fluidounces). I rarely employ *disinfectants;* in particular care should be exercised in the use of carbolic acid and mercuric chlorid, as dangerous intoxication may readily be induced thereby.

In obstinate cases of chronic inflammation of the bladder incision of the bladder—urocystotomy—has even been resorted to, with direct treatment of the wall of the bladder, and recently curettage of the mucous membrane. In the presence of chronic catarrh of the bladder courses of treatment at the **springs** have also been followed. Waters containing calcium sulphate have been particularly recommended, such as Carlsbad, Vichy, Ems, Neuenahr, Contrexeville, Wildungen. Also indifferent thermal springs (Pfäffers, Ragaz, Wildbad, Gastein) are frequently useful. If *retention of urine* occur in the course of inflammation of the bladder, the urine should be withdrawn by means of a catheter. Also in cases of *eccentric hypertrophy of the bladder* catheterization should be practised systematically in order to evacuate the bladder completely from time to time. In cases of *concentric hypertrophy of the bladder* the patient should be urged to retain the urine for as long a time as possible, in order to effect gradual distention of the viscus. Marked vesical tenesmus can be effectually relieved by a subcutaneous injection of morphin or by suppositories of morphin:

℞ Morphin hydrochlorate, 0.1 (1½ grains);
Cocoa-butter, sufficient to make 10 suppositories.
Dose: 1 suppository once or twice daily.

CARCINOMA OF THE URINARY BLADDER.

Etiology.—Carcinoma of the urinary bladder is generally **secondary**, and arises frequently by direct extension from adjacent organs (rectum, prostate gland, uterus). The disorder is, on the whole, an uncommon one, and it almost always occurs after the thirtieth year of life, and, as experience has shown, more commonly in women than in men.

Anatomic Alterations.—Carcinoma of the bladder is most commonly seated at the neck and at the base of the bladder. At times the morbid process consists in extensive carcinomatous infiltration of the wall of the bladder, particularly of the submucosa, while at other times it gives rise to a circumscribed neoplasm, attached to the interior of the bladder by means either of a broad base or of a slender pedicle. The new-growth may be an epithelial carcinoma (cancroid), or a villous carcinoma, or a scirrhus; least commonly it appears as a medullary carcinoma or colloid carcinoma. Not rarely the carcinomatous tissue undergoes disintegration, with the development of a **carcinomatous ulcer.** As a result of this process almost all the new-growth may be destroyed, so that the nature of the neoplasm will at times be recognizable only from the carcinomatous infiltration of its margins. Under such circumstances *rupture of the bladder* into adjacent organs (rectum, vagina, abdominal wall, perineum) may also take place. The *mucous membrane of the bladder* is almost always in a state of chronic inflammation.

Symptoms and Diagnosis.—Carcinoma of the bladder is often concealed for a long time behind the symptoms of *chronic cystitis*, and at times the true condition is disclosed only on post-mortem examination. Chronic cystitis should always arouse suspicion as to its carcinomatous nature if the symptoms of inflammation of the bladder appear to develop spontaneously in an elderly person, in the absence of signs of the presence of a calculus or of prostatic hypertrophy, if pallor and emaciation increase rapidly, and the inguinal glands are indurated and enlarged in consequence of carcinomatous degeneration. Frequently **hematuria** occurs, and this may be repeated from time to time and accelerate in marked degree the pallor and the debility. At times the bleeding is so profuse and uncontrollable as to cause death from hemorrhage.

The diagnosis of carcinoma of the bladder will be positive only when undoubted masses of **carcinomatous tissue** can be demonstrated in the urinary sediment, or when the new-growth becomes accessible to palpation or inspection. On examination of the urinary sediment it will be important to learn whether an unusually large number of multinucleated epithelial cells are present, or whether perhaps macroscopically visible shreds and flocculi of carcinomatous tissue can be found in the urine. Only rarely will it be possible to feel a carcinomatous growth of the bladder as a nodular tumor through the abdominal walls. Most commonly it can be palpated on examination through the rectum or vagina, or by means of a catheter. In the last event, one must be prepared to detect an earthy scratching with the catheter, because not rarely the surface of the new-growth is covered with a crystalline sediment. At times portions of new-growth remain impacted in the fenestrum of the catheter, and these should be carefully examined microscopically. Rarely, in women portions of tumor are observed to project into the urethra. Recently an element of accuracy and certainty has been added to the diagnosis of diseases of the bladder through the introduction of illuminating apparatus into that viscus—*urocystoscopy*—which renders possible thorough examination of the interior of the bladder.

Patients with carcinoma of the bladder often suffer from **severe pain in the hypogastric region**, which is prone to appear particularly during the night, and not rarely radiates for considerable distances (loins, thigh). At times **retention of urine** occurs, but **incontinence of urine** also may develop if the tumor has invaded the orifice of the urethra, and has destroyed the sphincter of the bladder. Febrile symptoms are not rarely present, particularly if the urine is greatly decomposed. Death generally occurs within a year as the result of exhaustion or urinary septicemia or rupture of the bladder, and at times also of uncontrollable hemorrhage. If the orifices of the ureters are obstructed by the new-growth, hydronephrosis, pyelonephritis, pyonephrosis, and septicemia may readily develop.

Prognosis and Treatment.—The prognosis of carcinoma of the bladder is still extremely grave, although it has been rendered rather more favorable in recent years since successful attempts have been made to remove the new-growth by **operative means.** No result can be effected by internal remedies.

PARASITES OF THE URINARY BLADDER.

Among **animal parasites** the presence of a free *echinococcus-cyst* in the bladder has been recorded in one case. In the Orient, particularly in Egypt, the *Distoma hæmatobium*[1] occurs not rarely in the blood-vessels of the bladder, and it may give rise to severe inflammation, ulceration, and hemorrhage from the mucous membrane. The disease can be diagnosed from the presence of distoma-ova in the urinary sediment, and these will be recognized from their oval shape and the lateral process. If the bladder and the intestine are adherent and in free communication, *ascarides* may at times gain entrance into the bladder, and can sometimes be removed from the urethra with the fingers.

Vegetable parasites, particularly bacteria, are, as has been mentioned, of great significance in connection with decomposition of urine and inflammation of the bladder. Fragments of *leptothrix* have been found in saccharine urine. At times special *sarcinæ* have been found in the urine. These are somewhat smaller than the sarcinæ of the stomach, but similar in their quadrangular shape and in their arrangement in groups of four or more. At times the sarcinæ form a considerable sediment. They occur in alkaline and in acid urine, and give rise to no particular symptoms. Some patients suffer from *bacteriuria*, their urine containing bacteria at the time of evacuation, presenting a light-yellow and turbid appearance, and being characterized by a stale odor suggestive of meat-broth. Symptoms are usually wanting. The condition is often obstinate. The administration of salol (1.0—15 grains—1 powder every two hours) may be recommended.

FOREIGN BODIES IN THE URINARY BLADDER.

Foreign bodies may be introduced into the bladder from without or find their way into the viscus from adjacent organs. Thus, in a case of a vesico-intestinal fistula **striated muscular fibers, vegetable cells,** and other **articles of food** have been found in the urinary sediment. At times flatus escapes from the bladder. Occasionally **gall-stones** are present in the urinary bladder, and require removal by means of cystotomy, having made their way into the bladder through the biliary passages or the intestine. **Hair** has also been observed in the urine—*pilimictio*—when communication has been established between the bladder and a dermoid or an ovarian cyst. Even bits of cartilage and of bone and teeth have been found in the urine under such circumstances.

NEUROSES OF THE URINARY BLADDER.

NOCTURNAL ENURESIS.

Symptoms and Diagnosis.—Nocturnal enuresis consists in unconscious evacuation of the urine into the bed during profound sleep, and generally in the first two hours, with the discovery only in the morning on awakening, from the wet body-linen and bed-linen, of what has taken place during the night. Some patients dream of standing before a trench or a pool of water and of having been commanded to pass urine; they obey, awake from the dream, and become conscious of having passed urine into the bed. In some cases the occurrence takes place only at long intervals, and particularly when considerable fluid has been ingested at night and the evening meal has been taken late. Other patients, however, wet the bed almost nightly. The patients often present a **pallid appearance** and an **excitable nervous manner.** In the majority of cases the disorder is curable. Generally it disappears at the latest at about the twelfth year of life, and only in the small minority does it persist throughout life. Under the conditions last named nocturnal may be complicated by *diurnal enuresis,* so that the clothing is wet even during the day and in the waking state, particularly on laughing, coughing, or making expulsive effort. This disorder exerts an unfavorable influence particularly upon the *mental state* of the patient, who avoids others and gradually withdraws from human intercourse, becomes hypochondriacal, and sometimes commits suicide.

Etiology.—Nocturnal enuresis is a **disease of childhood,** and if it occur, contrary to rule, in an adult, it is indicative of profound nervous disease (cerebral softening, general paralysis of the insane). At times the disorder is the result of **defective education.** It should be borne in mind that nocturnal enuresis is a natural condition in earliest childhood, and that it is corrected by training, particularly by regularly waking the child from sleep and insisting upon evacuation of the bladder. If the guardian is indifferent or careless, the child may readily become confirmed in the habit of nocturnal enuresis. In some cases **indolence** and **convenience** are responsible for nocturnal enuresis; the child is awakened from sleep by a desire to pass urine, but is disinclined to leave its warm bed for the purpose, and endeavors to suppress the act till morning, and is overtaken during sleep by the discharge of urine. An **inappropriate evening meal** also may give rise to nocturnal enuresis, especially if taken too late or in too large an amount, or if it contain too much liquid, particularly beer. I have observed nocturnal enuresis in a number of instances after indulgence in considerable amounts of fruit. It is noteworthy that **diabetes mellitus** and **contracted kidney** in children are at times first disclosed by nocturnal incontinence of urine, obviously in consequence of over-distention of the bladder with urine. At times nocturnal enuresis occurs as a result of **reflex irritation**, as, for instance, from the presence of intestinal worms, vesical calculi, phimosis, villous hyperplasia of the urethra, and adenoid vegetations in the bladder. Without doubt, **central nervous influences** are at times operative. It has been observed that the children of epileptics not rarely suffer from nocturnal enuresis. The condition has also been observed on a large scale in foundling asylums, in consequence of **imitation.** Girls are believed to suffer less commonly than *boys* on account of the greater capacity of the female bladder.

Prognosis.—As a rule, nocturnal enuresis is a curable disorder, and never dangerous to life, so that the prognosis is not unfavorable.

Treatment.—In every case of nocturnal enuresis the treatment should be begun by regulation of the **diet** and by **training.** The patients should receive their evening meal not less than two hours before going to bed, and much liquid, potatoes, and heavy articles of food in general should be avoided. The children should sleep upon a firm mattress, under a light covering, and should lie upon the side, because nocturnal incontinence of urine occurs with particular ease and frequency in the recumbent posture. It is important that the pelvis be elevated, and this may be accomplished by raising the foot of the bed. At ten, twelve, and three o'clock at night the child should be regularly awakened from sleep and made to pass urine, and this rule should not be abandoned until the nocturnal enuresis has remained permanently in abeyance. In addition, **causative conditions** should be relieved—**causal therapy.** Intestinal worms should be expelled, phimosis and villous growths of the urethra should be removed, and also hyperplasias in the nose. For anemic persons iron should be prescribed; for nervous patients, nervines, cold frictions, and the like. There have been recommended, further, **narcotics, tincture of cantharides, antipyrin, vesicatories** to the sacrum, the **electric current, massage, dilatation of the urethra with bougies, cauterization of the neck of the bladder, introduction of a cold sound** (*psychrophore*), **compresses for**

the urethra, closure of the urethra and the **preputial orifice with collodion** at night, in order that the patient shall be awakened from sleep when the evacuation of urine begins. In one case speedy success was obtained by means of **hypnosis and suggestion.**

HYPERESTHESIA OF THE URINARY BLADDER.

Etiology.—Hyperesthesia of the urinary bladder occurs principally in *hysterical, hypochondriacal*, or *nervous individuals*, in whom the disorder is often due to mental over-exertion, alcoholic and venereal excesses, excessive indulgence in tobacco, masturbation, and the like.

Symptoms and Diagnosis.—The patients complain of intense vesical tenesmus, which is appreciated when the bladder contains only a small amount of urine. This tenesmus must be promptly responded to in order to avoid severe pain, or suppression of urine if the delay be too protracted. Some patients withdraw from human society and prefer to be alone, because they are free from vesical tenesmus for scarcely half an hour. The urine is frequently unaltered, although at times it is highly acid.

Prognosis and Treatment.—The prognosis is not unfavorable if the clinician is capable of exerting a favorable mental influence upon the patient through his personality. The patient should, in the first place, be assured that the disorder is a purely nervous one, without anatomic alteration ; and he should be advised to take little liquid, and if possible to avoid alcoholics entirely, and should be urged not to respond to the desire for micturition at once, but gradually to make the intervals between evacuation longer and longer. In addition, the primary disorder should receive the usual treatment.

SPASM OF THE URINARY BLADDER (CYSTOSPASM).

Etiology.—Spasm of the muscular coat of the urinary bladder may result from anatomic alterations (disease of the bladder, the spinal cord, or the brain), or it may be a purely nervous disturbance. Only the latter will be considered in the following description. The condition occurs most commonly in connection with **hysteria, hypochondriasis,** and **neurasthenia. Masturbators** at times suffer from spasm of the urinary bladder. Occasionally the disorder arises as a result of **reflex irritation,** as, for instance, in consequence of disease of the uterus or the ovaries, the presence of worms in the intestine, etc. At times it occurs after the ingestion of young wine or beer, or of asparagus.

Symptoms and Diagnosis.—Spasm of the muscular coat of the bladder may involve the detrusor or the sphincter, or both muscular structures together. **Spasm of the detrusor of the bladder**

is manifested by abnormal desire for micturition, which may appear even after the accumulation of only a small amount of urine in the viscus. The disorder may readily be mistaken for hyperesthesia of the bladder; but the latter is a permanent condition, while spasmodic states of the detrusor occur only periodically, and urinary difficulties are wanting in the intervals. **Spasm of the sphincter of the bladder** is attended with disturbances in evacuation of the urine. In spite of existing tenesmus, either the urine is voided drop by drop—**spastic dysuria**—or there is complete retention of urine. If a catheter be introduced into the bladder, it will encounter resistance at the orifice of the viscus before it can enter its cavity. The patients complain of intense pain in micturition, and which not rarely radiates to remote parts (anus, testicle, glans penis). **Simultaneous spasm of the detrusor and the sphincter of the bladder** causes abnormally increased desire for micturition, and obstruction and pain in the evacuation of urine. The former is due to spasm of the detrusor, the latter to that of the sphincter. The pains are frequently so severe that the patients become pale, appear bathed in perspiration, and may even faint. Nervous spasm of the bladder is distinguished from spasm of the bladder dependent upon anatomic alterations by the unchanged condition of the urine, and the absence of demonstrable lesions in the bladder and other organs.

Prognosis and Treatment.—Spasm of the bladder is an annoying disorder rather than dangerous to life. A **warm bath** (30° R.—37.5° C.—99.5° F.) should be prescribed, and the patient urged to pass water in the bath. In addition, **hot cataplasms** should be applied over the bladder, and **morphin** should be injected subcutaneously or employed in suppositories. In order to prevent recurrence of the spasm it will be necessary to adopt **causal treatment** and relieve the underlying disease.

PARALYSIS OF THE BLADDER (CYSTOPLEGIA).

Etiology.—Paralysis of the bladder is a frequent symptom of many **diseases of the spinal cord**, as, for instance, myelitis, transverse myelitis, tabes dorsalis, multiple sclerosis, and spinal concussion. **Diseases of the brain** (softening) also are not rarely attended with paralysis of the bladder. The paralysis of the bladder resulting in connection with opium-poisoning may be designated **toxic**. At times paralysis of the bladder develops in the sequence of **excessive distention** of the viscus. Such a condition may occur in comatose persons, who are unconscious of the stimulus to evacuate the bladder, so that the organ may become filled with a large amount of urine, as, for instance, in cases of typhoid fever, meningitis, and the like. Persons also whose occupation renders it difficult to respond promptly to the desire for micturition (speakers, teachers, actors, officers) may acquire paralysis of the bladder as the result of frequent over-distention. Cystoplegia occurs not rarely in persons suffering from stricture of the urethra or prostatic hypertrophy, because also both of these conditions give rise to distention of the bladder. At times paralysis of the bladder is dependent upon **diseases of the viscus,** as, for instance, inflammation or carcinoma. It may occur as a purely **nervous disorder** in hysterical, hypochondriacal, and nervous persons. It develops at times in association with **debilitated states** of the body, as, for instance, after onanism and senile marasmus.

Symptoms and Diagnosis.—In the same way as with spasm of the bladder, paralysis of the bladder at times involves the detrusor, at other times the sphincter, or at still other times both muscles together. *Paralysis of the detrusor of the bladder* is attended with difficulty in the evacuation of urine. The patient must make strong expulsive efforts in order to expel the urine, which is discharged in a less powerful stream than normally. The greater the degree of paralysis, the more nearly does the evacuation approach gradual dribbling. The bladder at times becomes so greatly filled with urine that it may rise to the level of the umbilicus, and even above, and the resulting excessive stretching of the detrusor is well adapted to augment the paralysis. Eventually the power of voluntary evacuation of the urine is lost, and the patient is condemned to systematic resort to the use of the catheter. *Paralysis of the sphincter of the bladder* is attended with **dribbling of urine—incontinence**—which often is at first manifested by the escape readily of urine into the clothing on laughing, coughing, or making expulsive efforts. Not rarely bacteria gain access to the bladder when the orifice of the viscus is open, with decomposition of the urine and inflammation of the bladder. Simultaneous *paralysis of the detrusor and the sphincter of the bladder* gives rise to a combination of the two groups of symptoms described. The urine escapes involuntarily from the bladder until its level falls just below the vesical orifice of the urethra. If a catheter be introduced into the bladder immediately after the evacuation of urine, additional urine will be evacuated which had remained at the base of the bladder, and is therefore designated *residual urine.* Paralysis of the muscular coat of the bladder is distinguished from spasm of the musculature of the bladder particularly by the absence of pain. The *duration of paralysis of the bladder* depends upon the causative factors in the individual case.

Prognosis.—Whether paralysis of the bladder is curable or not depends mainly upon the causative factors. If these be incurable, the principal danger consists in the readiness with which decomposition of the urine takes place and the frequently complicating *inflammation of the bladder,* and which in turn may give rise to pyelonephritis and urinary septicemia.

Treatment.—In the presence of paralysis of the bladder attention should be given to **systematic evacuation of the viscus.** At times this end can be attained by pressure upon the bladder through the abdominal wall; otherwise resort must be had to the introduction of a catheter, which naturally must be carefully sterilized. Artificial evacuation of the urine should be practised thrice daily. In order to *strengthen the muscular coat of the bladder* cold frictions should be applied over the bladder, subcutaneous injections of strychnin or of ergotin made, and the galvanic or the faradic current employed. In addition, careful attention should be given to the treatment of the causative conditions—**causal therapy.**

IV. DISEASES OF THE MALE SEXUAL ORGANS.

IMPOTENCE IN THE MALE.

Etiology.—Inability to effect coitus successfully is designated impotence in the male. At times the condition is dependent in a purely mechanical manner upon **diseases of the penis,** which prevent the introduction of the male organ into the vagina. The penis may be unduly short, or it may be rendered so from the presence of marked hydrocele or of scrotal hernia. Angular distortion of the penis in consequence of cicatrices or new-growths may cause impotence. **Diseases of the testicles** may give rise to impotence if the secretion of seminal fluid is abolished, for, as a rule, erection takes place only when the testicles secrete semen. At times impotence in the male results from **wasting discharges** and **exhaustion,** and develops in the sequence of severe diseases, such as contracted kidney, diabetes mellitus, onanism, and venereal excesses. **Toxic impotence** may occur as a result of the protracted use of potassium bromid, camphor, lupulin, arsenic, salicylic acid, and morphin. At times impotence in the male is caused by **nervous influences.** Frequently it occurs in the course of *tabes dorsalis. Neurasthenia* also is not rarely attended with impotence. **Psychic impotence** is particularly to be mentioned. This results from a fear on the part of the patient that he is not potent, and this paralyzing dread either prevents entirely erection of the penis or permits but transient erection, or there occur premature discharge of semen and rapid flaccidity of the penis. Psychic impotence occurs at times in chaste persons, particularly on the first attempts at sexual intercourse. It occurs also in masturbators, in whom a guilty conscience exerts a paralyzing influence as soon as an attempt at normal sexual intercourse is made. Also, the dissolute

é of the brothel is at times attacked with psychic impotence
ual intercourse with his wife. Psychic impotence may be
ested only in sexual relations with certain women. Some men
pable of overcoming this by certain devices which are mostly
sive to a healthy mind. Psychic impotence particularly has an
ious influence upon the mental state, and often induces suicide.
mpotence in the male may be *temporary* or *permanent*, accord-
y as the causative factors are curable or irremediable.

he **prognosis** and the **treatment** also depend upon the
ure of the cause. In the presence of neurasthenia, in addition
nervines, courses of treatment with **cold water**, in the mountains
at the seaside, will be necessary. In cases of psychic impotence
e patient should by intelligent encouragement be imbued with
nfidence in his potence. Too frequent sexual intercourse and
tempts at intercourse during alcoholic intoxication should par-
icularly be avoided. Under such conditions also cold sponging of
he hypogastrium and a sojourn in the mountains or at the seaside
re often to be recommended. At times good results are secured
from **hypnosis** and **suggestion**. Upon the latter probably depend the favorable effects reported from the employment of tablets of testicular tissue and injections of testicular fluid or of sperm. Applications of the galvanic and of the faradic current to the lumbar spine and to the sexual organs have also been made.

STERILITY IN THE MALE.

Sterility in the male is present if in spite of natural methods of coitus no seminal fluid is discharged into the vagina of the woman—so-called *aspermatism;* or if the seminal fluid introduced does not contain fructifying spermatozoids—*azoöspermia.*

Aspermatism is most commonly due to obstruction of the seminal ducts; less commonly it is caused by nervous disturbances. Aspermatism is often dependent upon *stricture of the urethra* in consequence of chronic gonorrhea. *Disease of the prostate gland* also not rarely causes aspermatism, as the enlarged gland may compress the urethra and thereby diminish its lumen, or as a result of contracting changes may give rise to such a deflection that the semen when ejaculated will not escape anteriorly, but posteriorly into the bladder. *Disease of the seminal vesicles* may cause aspermatism by exerting pressure upon and diminishing the lumen of the ejaculatory ducts. At times *phimosis* is responsible for aspermatism if the urethra is, as a result, materially constricted. *Injuries to the perineum* are also capable of causing aspermatism through compression of the ejaculatory ducts. Occasionally the condition has been observed after operations for stone, if the ejaculatory ducts have been cut by the surgeon's knife.

[illegible] *rmatism* occurs principally in neurasthenic individuals; coitus can be effected without difficulty, but ejaculation may not take place or only in intercourse with certain women, but not with others. This form of aspermatism occurs especially in masturbators and in men who have engaged excessively in illegitimate sexual intercourse. Under such circumstances it may happen that ejaculation of seminal fluid fails to take place, especially in sexual intercourse with the wife. A variety of *physiologic aspermatism* occurs in healthy men who indulge in sexual intercourse at too short intervals.

Aspermatism is readily recognizable from the fact that in sexual intercourse ejaculation of seminal fluid does not take place at all, or occurs only after the penis has become flaccid and has been withdrawn from the vagina. Should an ejaculation have taken place, but into the bladder, turbid urine containing many spermatozoids will be evacuated after the act of coitus.

The *duration* and the *prognosis*, as well as the *treatment* of aspermatism vary in accordance with the causative factors. In cases of psychic aspermatism an endeavor should be made to improve the general condition, while the patient should be encouraged with assurance, nervines be administered, courses of treatment with cold water, in the mountains and at the seaside, be pursued, and applications of electricity be made.

Azoospermia is characterized by an absence of the secretion of the testicles, and therefore of fructifying spermatozoids, although in the sexual act secretion is discharged from the seminal vesicles and the prostate gland. The condition can be readily recognized on microscopic examination. The ejaculated fluid possesses the so-called seminal or spermatic odor, because this is due not to the secretion of the testicles—the true seminal fluid—but to that of the prostate gland. Azoöspermia is a most common disorder. Most sterile marriages are dependent upon azoöspermia, and not upon the often insignificant displacements and other abnormalities of the unoffending wife. *Gonorrheal urethritis* is responsible for azoöspermia with especial frequency if, in conjunction with gonorrhea, bilateral orchitis or inflammation of the vasa deferentia develops, preventing the entrance of seminal fluid into the seminal vesicles and ejaculatory ducts. The condition is generally incurable.

Spermatozoids will naturally be absent from the secretion of the generative organs if the tissues of the *testicles* are seriously diseased and the normal function of the latter is suppressed. Such conditions are generally attended with impotence, and are therefore not included in azoöspermia.

INVOLUNTARY DISCHARGE OF SEMINAL FLUID (SPERMATORRHEA).

Spermatorrhea consists in the discharge of secretion from the generative glands independently of the sexual act. The ejacuated fluid may be derived from the testicles, the prostate gland, the seminal vesicles, the glands of Littre or those of Cowper.

TRUE SPERMATORRHEA.

Symptoms and Diagnosis.—True spermatorrhea is attended with the discharge from the urethra of testicular secretion, which s readily recognized on microscopic examination from the presence of numerous spermatozoids. Occasionally these are imperfectly developed, with a slender filament attached to the head. Their want of motility also is at times conspicuous. At times so-called seminal cells—large multinucleated cells—are present. If seminal fluid in considerable amount is admixed with the urine, the latter acquires a milky, fat-like appearance—**lipuria**—which disappears when the urine is agitated with ether, preferably after addition of potassium hydroxid.

Involuntary discharge of seminal fluid occurs at night from time to time in every healthy man—**nocturnal pollution.** Under such conditions lascivious dreams generally occur, and with customary erection of the penis and a pleasurable sensation a discharge of seminal fluid takes place, and, as a rule, causes awakening. The occurrence is usually followed by a sense of great freshness on the following night. Such pollutions are repeated in different men at varying intervals, in some within from 4 to 8 weeks, in others within from 4 to 8 days, in accordance with the temperament, the constitution, and the mode of life of the individual.

Abnormal nocturnal pollutions are characterized by the recurrence of seminal emissions during the same night or on several successive nights, by incomplete erection of the penis or its total absence, by the absence of voluptuous dreams, by failure to be awakened by the discharge, and the failure of the latter to be followed by a sense of freshness, but by one of exhaustion, with the association of headache, vertigo, palpitation of the heart, a sense of constriction and of weakness in the legs.

A more profound degree of the disorder is attended with seminal discharges also during the day and the waking hours—diurnal pollutions. These may occur upon any sexual excitement, however slight, as, for instance, in reading obscene books, in looking at voluptuous pictures, in contact with women, and even in looking at women or in thinking of sexual matters. This condition may eventually lead to *permanent spermatorrhea,* which is, however, a rare disorder. The latter is attended with the constant discharge of seminal fluid from the urethra and its accumulation in the preputial sac without any pleasurable sensation. Often this condition is preceded for a time by *spermatorrhea attending micturition and defecation,* the discharge of seminal fluid taking place only in association with those acts.

Spermatorrhea exerts a most injurious *psychic influence* upon the patient, and the more so as it is frequently observed in persons with an abnormally irritable and excited nervous system. The patients are worried particularly by the thought that they can never again be potent, and in many cases there is present in addition the consciousness that the patient is himself responsible for the disorder as a result of sexual excesses and masturbation. The great fear of a gloomy future on the one hand, and the want of courage to renounce sexual abuse forever on the other hand, are often remarkable. Persons with spermatorrhea may for a long time preserve a healthy and even a vigorous appearance; but, as a rule, they gradually grow paler and more emaciated, are troubled with sleeplessness and increasing exhaustion, become more and more neurasthenic, and some reach the insane asylum or commit suicide.

Etiology.—Spermatorrhea is a common sequel of **sexual excesses,** both natural and unnatural—onanism. At times it occurs as a result of **reflex irritation** in the presence of gonorrheal urethritis, phimosis, retained preputial sebum, worms, hemorrhoids, anal fissure, and the like. Occasionally the disorder is dependent upon **disease of the central nervous system,** as, for instance, tabes dorsalis or a transverse lesion of the spinal cord. Spermatorrhea occurs also at times during epileptic convulsions. In some cases spermatorrhea is induced by **conditions of general debility.** It may therefore be observed in the sequence or course of typhoid fever, pulmonary tuberculosis, and diabetes. Members of families with an inherited nervous temperament and persons who have made themselves nervous by excessive mental activity or by excessive indulgence in alcohol, tea, or tobacco are undeniably predisposed to spermatorrhea.

The **prognosis** and the **treatment** depend primarily upon the causative factors. Onanism and sexual excesses are to be avoided, and irritative conditions (gonorrhea, phimosis, worms, etc.) to be relieved. To subdue excessive sexual desire, **anaphrodisiacs** have been recommended (potassium bromid—3.0—45 grains—at night in a wine-glassful of milk; camphor or lupulin—0.3—4½ grains—four times daily). The **mode of life** is important. The evening meal should be light, and be taken three hours before bedtime. The bed should consist of a hard mattress and a light cover. The patient should try to sleep on his side, as experience has shown that pollutions readily occur in the dorsal decubitus. An effort should be made to make a moral impression on the patient, and to invigorate his general condition by cold baths or spongings, systematic walks, and in the summer by a sojourn in the mountains or at the seaside.

The treatment of spermatorrhea has been largely undertaken by *charlatans.* As a rule, a circular is first sent the patient arousing his fears, and finally a useless medicinal remedy or some form of apparatus is offered at an extravagant price. Thus, *alarms* have been constructed for the purpose [illegible] of an impending pollutio[illegible] [illegible] Lallemand, who attribut[illegible] [illegible] inflammation of the caput gallinagin[illegible] [illegible] made applications of silve[illegible] [illegible] application of a *cold bougie* or [illegible]

[illegible]RRHEA.

[illegible] discharge from the urethra [illegible] spermatic odor, and o[illegible] [illegible] cylindric epithelium, lamin-[illegible] [illegible] granules or scales of yellowis[illegible] [illegible] *crystals* is especially charac-[illegible] [illegible] resemble asthma-crystals, bu[illegible] [illegible] acuole pyramids. These crys-[illegible] [illegible] 1 per cent. solution of ammo[illegible] [illegible]. Prostatorrhea may occur [illegible] processes in the prostatic gland, [illegible] [illegible]ence of gonorrhea or anemia, but [illegible] [illegible] of the prostate. The pros-[illegible] [illegible] not rarely after defecation, at [illegible] efforts, or it is discharged exter-[illegible] [illegible] the rectum and pressure is made [illegible] anterior wall of the bowel. The [illegible] [illegible]necessary fear on the part of the [illegible] [illegible] is mistaken for seminal fluid, but [illegible] [illegible] danger.

[illegible] can be recognized from the presence of [illegible] and Trousseau, which resemble swollen [illegible]bulin-substance. The glands of Cowper [illegible] secrete considerable fluid on erection. The [illegible] cells and epithelial cells in small number, [illegible]ation of the lips of the urethral meatus.

[illegible] OF THE ADRENAL BODIES.

ADDISON'S DISEASE.

[illegible] and Diagnosis.—Addison's disease is attended [illegible] **[illegible]coloration of the skin and the mucous membranes** [illegible] progressive **asthenia.** At times the discoloration [illegible] the mucous membranes is the earliest symptom, [illegible] times anorexia, vomiting, irregularity in movement [illegible] general malaise, and the like, have been present [illegible] time previously as **prodromes.** These prodromal symp-[illegible] perhaps only the results of auto-intoxication dependent upon disease of the adrenal bodies. The cutaneous alterations generally appear earliest in parts of the body exposed to the light; then follow places subjected to pressure, or under normal conditions the seat of pigmentation. Eventually the discoloration involves the skin of the entire body. The dark color of the skin, which often is suggestive of the appearance of bronze, has helped to give the disease the name of *bronze-disease*. At the commencement of the discoloration of the skin small, dark spots appear; these soon increase in size, coalesce with adjacent spots, and then discolor the skin uniformly throughout a considerable extent. The first alterations are usually observed in the face, which acquires a brownish hue, as if the skin had been burned by the rays of the sun; subsequently the color becomes more like that of graphite, so that it may be confounded by the inexperienced clinician with cyanosis. The patient resembles a mulatto. Also, the hands and the forearms, in persons who go barefooted the dorsum of the feet and the legs, participate early in the cutaneous discoloration. The palmar aspect of the hands and the plantar aspect of the feet, however, as well as the nails of the fingers and the toes, remain unaltered, and are often conspicuous for their bright and pale appearance. On the trunk a dark, discolored cutaneous girdle is observed to encircle the junction of the abdomen and the thorax, corresponding to the constriction induced by waist-bands and other articles of clothing. The nipples, the axillary cavity, the scrotum, and the penis are usually conspicuous for their dark-brown or black color. Of the *mucous membranes*, that of the **oropharynx** becomes affected particularly early, and especially upon the mucous membrane of the cheeks opposite the teeth a brownish or blackish oval or irregular area is frequently found whose borders often pursue an irregular, serrated course. Now and then, but on the whole uncommonly, small brownish or blackish spots are observed upon the **conjunctiva**, which may readily escape detection on account of their small size. In women similar alterations occur upon the **mucous membrane of the labia minora and the vagina.**

Addison's disease is readily recognized from the discoloration of the skin and the mucous membranes. Long-continued employment of silver nitrate may give rise to a dark discoloration of the skin—so-called **argyria** or **argyrosis**; but the fact of the internal or external employment of this agent can be determined by inquiry into the history. The same statement is applicable to **arsenical melanosis**; that is, that dark discoloration of the skin that appears in some persons after long-continued use of arsenic. The color of the skin in cases of Addison's disease differs from that observed in **brunets** and in those who live in the **tropics** in the fact that with the former general symptoms are present besides, and these will shortly be described. It is noteworthy, further, that in some healthy persons dark spots like those of Addison's disease occur upon the mucous membrane of the lips or the cheeks. Persons who have been infested for a long time with **body-lice**—*Pediculi vestimentorum*—often present a brownish or blackish discoloration of the skin in consequence of extravasation of blood at the points of biting—**cutaneous nigrities**—but the face and the mucous membranes escape. In persons of uncleanly habits the skin may present a brownish or blackish appearance due to **dirt**: but this can readily be removed by means of soap and water.

Constitutional symptoms are generally not long absent. There is progressive loss of appetite, with nausea and vomiting and progressive asthenia. Often debilitating diarrhea sets in. At times complaint is made of pain in one or both renal regions, and this often is increased on pressure. Also, pains in muscles and joints occur frequently. The action of the heart is generally accelerated, and the tension of the pulse is greatly diminished. The mental state is generally one of depression and dejection. At times vertigo, a sense of pressure in the head, tremor, convulsions, and paralysis occur. As a rule, the patients fail gradually with progressive asthenia. In a number of instances I have observed the rather sudden development of coma, followed by death, the condition suggesting auto-intoxication. It is noteworthy that at times the constitutional symptoms and the comatose condition described may occur in the absence of discoloration of the skin and the mucous membranes, whereas an autopsy may disclose only disease of the adrenal bodies, thus auto-intoxication without bronzing. The duration of the disease extends at times over more than a year.

Anatomic Alterations.—The dark **discoloration of the skin and the mucous membranes** persists after death. On microscopic examination of the skin brownish or blackish pigment-granules are found in the lowermost layers of cells of the rete Malpighii. Round and branched cells filled with pigment-granules occur also in the cutis. Of the internal organs, the **adrenal bodies** particularly attract attention, being diseased in the majority of cases, though not in all. Most commonly the condition is one of tuberculous-caseous change, which in turn may be primary or secondary; less commonly there may be carcinoma, amyloid degeneration, gummatous nodules, hemorrhage, chronic fibrous inflammation, or even echinococcus. At times the alterations are only unilateral, but in most cases they are bilateral. In addition to the alterations in the adrenal bodies, degeneration of ganglion-cells and nerve-fibers and connective-tissue hyperplasia in the **solar plexus** have been observed; and because in some cases of Addison's disease the adrenal bodies have appeared unchanged the opinion has been formed that the disorder is dependent upon disease of the solar plexus of the sympathetic system, and that the alterations in the adrenal bodies are rather subordinate. Naturally, this view is not in accord with the fact that even expert observers have found the solar plexus intact. There maining organs exhibit in part signs of **emaciation.** In cases of secondary tuberculosis of the adrenal bodies the same condition has been found in the lungs, the kidneys, the bones, and elsewhere.

Etiology.—The causes of Addison's disease are often unknown, and the condition must then be designated *cryptogenetic.* At other times **tuberculosis or carcinoma in other organs,** antecedent **syphilis, suppuration,** or other **wasting discharge,** may suggest that the adrenal bodies are the seat of tuberculosis, carcinoma, gumma, or amyloid disease, with consequent development of Addison's disease. At times the disorder is attributed to **injuries in the renal region,** and it is dependent upon hemorrhage. Generally *adults* are attacked by the disease, which is the more common in *men.*

The pigment in the skin and the mucous membranes is derived from the transformation of hemoglobin, but the reason for the destruction of red blood-corpuscles in Addison's disease is unknown. It has been thought that in consequence of disease of the adrenal bodies certain substances are retained in the blood (pyrocatechin? taurocholic acid?), which cause marked destruction of red blood-corpuscles. In those cases in which the adrenal bodies are found unaltered it would be necessary to assume that the diseased abdominal sympathetic nerve exerted the same influence upon the activity of the adrenal bodies as if the adrenals themselves were diseased. Where the destruction of red blood-corpuscles and where the transformation of the hemoglobin takes place, and why the pigment is deposited precisely in the skin and the mucous membranes, are all questions to which at present satisfactory answers cannot be given.

Prognosis.—The prognosis of Addison's disease is unfavorable, as the disorder invariably terminates fatally.

Treatment.—*Specific remedies* for Addison's disease are not known. Of late, **organotherapy** has been attempted, and tablets of adrenal extract have been administered; but the reports as to the results are contradictory and still too scanty to permit of a final opinion in one or the other direction. There thus remains scarcely any but **symptomatic treatment.** An endeavor should be made particularly to maintain, and if possible to improve, the general condition by means of nutritious food, codliver-oil, and preparations of iron or iodin. In the presence of antecedent syphilis the employment of mercurials and iodids would be included in the **causal therapy.**

PART V.

DISEASES OF THE NERVOUS SYSTEM.

I. DISEASES OF THE PERIPHERAL NERVES.

PERIPHERAL PARALYSIS.

PRELIMINARY CONSIDERATIONS.

PERIPHERAL paralysis frequently comes under observation of the practitioner for treatment. Among the **causative factors exposure to cold, traumatism, toxic influences**, and **infections** may be mentioned, and, accordingly, a distinction has been made between refrigeratory (rheumatic), traumatic, toxic, and infectious peripheral paralysis. Toxic and infectious influences exhibit allied relations with each other in so far as infectious paralysis also is due to poisons, which, however, are of bacterial origin (toxins). As experience has shown, different nerves are involved in peripheral paralysis with varying frequency. The nearer the surface and the longer the superficial course of a nerve, the more is it exposed particularly to the action of cold and traumatism, and the more frequently is it the seat of paralysis. Paralysis of the facial and of the radial nerve is, therefore, of common occurrence. In cases of toxic paralysis the remarkable and hitherto unexplained phenomenon is observed that definite nerves are generally paralyzed by certain poisons. Thus, it is well known that lead-poisoning peculiarly gives rise to bilateral radial paralysis. Peripheral paralysis can be readily recognized, for the paralyzed muscles are incapable of contraction and exerting their functional activity. In favor of the peripheral origin of a paralysis is the fact that only a few nerves and their related muscles are involved. Also, when all of the nerves and muscles of one extremity are paralyzed the condition is generally due to peripheral causes, which often are seated in the nerve-plexus controlling the paralyzed extremity. On the other hand, paralysis of both upper and lower extremities, or of all the lower extremities simultaneously—paraplegia—is indicative of a spinal origin, and paralysis of the arm and the leg upon one side of the body—hemiplegia—is indicative of a cerebral origin.

Great importance in the **diagnosis** and the **prognosis** of peripheral paralysis is attached to the **electric irritability** of the paralyzed nerves and muscles. If a nerve is injured anatomically in only a slight degree, if therefore the condition is one of *slight peripheral paralysis,* which, as experience has shown, generally subsides in from two to four weeks, the electric irritability of the affected nerves and muscles exhibits no alteration as compared with the healthy side, and both nerves and muscles react to the faradic and the galvanic current with equal current-strength as the corresponding muscles and nerves upon the healthy side. A *peripheral paralysis of moderate severity* subsides in the course of from four to eight weeks, and can be recognized from the presence of partial degenerative electric reaction. This is manifested by the circumstance that for the stimulation of the diseased nerve-trunk by means of the faradic or the galvanic current (so-called indirect stimulation) considerably stronger currents are required than for the corresponding nerves of the healthy side. On direct stimulation of the paralyzed muscles the irritability to the faradic current will be found diminished, while that to the galvanic current is increased, and, besides, in contradistinction from healthy muscles, a considerably feebler galvanic current is required to obtain anodal closure-contraction than kathodal closure-contraction. This condition is spoken of as a reversal of the normal contraction-formula, AnClC > KaClC.

A *severe peripheral paralysis* either requires from eight to twelve weeks, or even more, for recovery to take place; or only partial recovery takes place, the motility of only some muscles being restored, while others remain paralyzed; or no improvement whatever ensues, but, on the contrary, the paralyzed muscles undergo progressive atrophy, become permanently shortened (contracture), and are periodically involved in involuntary contraction. On electric examination of a severe peripheral paralysis **total degenerative electric reaction** will be found. As compared with partial, total degenerative reaction is distinguished by the fact that the paralyzed nerve has lost its irritability completely to both faradic and galvanic currents. The manifestations on direct irritation of the muscles by means of the faradic or the galvanic current agree with those of partial degenerative reaction. Should recovery from a severe peripheral paralysis take place, it is distinctive for voluntary muscular movement to occur earlier in the paralyzed parts than it is possible to induce it by electric stimulation of the previously paralyzed nerves. The **character of the muscular contractions** that take place in cases of severe and moderately severe paralysis upon stimulation with the galvanic current is further noteworthy. The muscular contractions are not short and lightning-like, but slow and protracted, and they resemble *peristaltic muscular movements.* This phenomenon is not less significant in the recognition of partial and total degenerative electric reaction than the reversal of the contraction-formula.

In connection with both partial and total degenerative electric reaction, not only is the direct irritability of the paralyzed muscles to the galvanic current increased, but also the **mechanical muscular irritability** is augmented, so that even gentle percussion of the muscles is sufficient to induce active contraction. In precisely the same way as with galvanic muscular stimulation, also with mechanical irritation, it will be found that the muscular contraction is not lightning-like, but slow and peristaltic.

Division of nerves in animals has demonstrated that the manifestations of total degenerative electric reaction are dependent upon degeneration of the nerve-fibers (destruction of the medullary sheath and the axis-cylinder, multiplication of the nuclei in the nerve-sheath of Schwann). In consequence, the diseased nerve is incapable of transmitting, and of being stimulated by, the faradic and the galvanic current. The paralyzed muscles also undergo anatomic alterations. The individual muscle-fibers become smaller, and exhibit multiplication of the nuclei of the sarcolemma and increase of the intermuscular connective tissue. Under such conditions the diseased muscle loses its power of responding with contraction to brief stimulation by the faradic current, while the irritability to the galvanic current is increased. The electric irritability of the diseased nerve can be restored only if the degenerated nerve-fiber is reformed or regenerated. The axis-cylinders develop first. In this stage of regeneration the nerve-fibers are already capable of conducting impulses of the will; but they become susceptible to electric stimuli only after the previously naked axis-cylinders are surrounded with a medullary sheath.

The phenomena of degenerative electric reaction are not at once present with the onset of peripheral paralysis, but develop gradually only in the course of the second week of the disease, as some time is required for the nerve and its related muscles to undergo degenerative atrophy.

Degeneration of peripheral nerves and muscles occurs not only in connection with diseases of peripheral nerve-trunks, but also when the large *motor-trophic ganglion-cells in the anterior horns of the spinal cord* are diseased. Disorders of the latter variety are designated also *anterior poliomyelitis.* Consequently, degenerative electric reaction is encountered also in the disease named. The ganglion-cell in the anterior horns of the spinal cord, its axis-cylinder process, and the distribution of the latter in the peripheral nerve-trunk to the related muscle form a continuous whole, or a *neuron.* The phenomena of degenerative electric reaction may be so interpreted as to indicate destruction of the spinal-peripheral neuron at its central point of origin or in the course of its peripheral distribution: in the first instance a degeneration of the whole neuron resulting, in the latter only of the peripheral portion beyond the focus of the disease. In the presence of disease of the ganglion-cells, accordingly, the phenomena of degenerative electric reaction will be present in the entire peripheral distribution of the nerve, and in the presence of disease of the peripheral nerve-trunk itself only in the distribution of that portion of the nerve distal to the situation of the disease-focus.

In the **treatment** *of peripheral paralysis* **causal therapy** should always be considered. When exposure to cold has been operative salicylic acid or sodium salicylate may be employed (1.0—15 grains—1 powder every two hours until ringing in the ears occurs). Diaphoretics (hot infusions, pilocarpin subcutaneously) and hot cataplasms, applications of cotton-wadding, and the like, have also been employed. Antecedent syphilis will require mercurial inunctions and administration of potassium iodid (5.0 : 200 —75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily). Potassium iodid also may be employed in the treatment of toxic paralysis, for in cases of lead-poisoning it has been shown that potassium iodid aids in the elimination of lead from the body. Causal treatment occupies a conspicuous place, further, in all cases of traumatic peripheral paralysis. If nerves are injured by compression, the injurious influence should be removed. Paralysis of the nerves of the arms, for instance, develops at times in consequence of pressure induced by improperly constructed or badly fitting crutches, and under such conditions these supports must be dispensed with for the time and be properly adjusted after recovery has taken place; or, after fractures of bone, nerves are sometimes surrounded by callus, and paralysis results from the pressure exerted. Under such conditions the only remedy consists in exposure of the callus by operation, its division with a chisel, and the setting free of the imprisoned nerve. At other times a nerve has been divided and paralyzed, and remains paralyzed, although the cut extremities of the nerve have united by means of a connective-tissue cicatrix. Under such conditions remarkably successful results have been at times obtained by excising the connective-tissue cicatrix and freshening the extremities of the nerve with the knife, and uniting them by means of suture.

Specific remedies in the treatment of peripheral paralysis are as yet unknown. It was formerly thought that the electric current was capable of accelerating the regeneration of diseased and degenerated nerve-fibers, but this is scarcely correct. Electric treatment also must be included among symptomatic measures. Also, in the purely **symptomatic treatment of peripheral paralysis** employ often is made of salicylic acid, sodium salicylate, and potassium iodid. In addition, **inunctions** of mercurial ointment, potassium-iodid ointment, veratrin-ointment, strychnin-ointment, alcoholic applications (camphorated spirit, spirit of formic acid, spirit of mustard, compound spirit of angelica, etc.), or subcutaneous **injections** of strychnin nitrate (0.1 : 10—1½ grains : 2½ fluidrams; from 0.25 to 0.5—4 to 8 minims—thrice daily) may be mentioned.

Mechanical treatment with massage of the paralyzed muscles is advantageous in order to stimulate the nutrition of the muscles as much as possible, as these readily undergo atrophy due to inactivity in consequence of lack of use. The results of *electric treat-*

ment also are scarcely more than mechanical, inasmuch as after electric contractions paralyzed muscles emaciate less rapidly. As long as paralyzed muscles respond to the faradic current this should be employed, and with a well-moistened and firmly applied electrode movable from place to place, while the other (indifferent) electrode, as large as possible, is applied to the sternum, the individual paralyzed muscles are stimulated daily to contract for from ten to twenty times. Care should be taken to employ currents just strong enough to cause contraction of the paralyzed muscles, as currents of too great strength may produce *electric contractures* in the paralyzed muscles, which may not subside and may give rise to unfortunate disfigurement if, for instance, the muscles of the face are involved. If the paralyzed muscles do not respond to the faradic current, the galvanic current should be employed, preferably by labile application: the anode (+, positive pole) is applied to the sternum, and the more actively stimulating kathode (—, negative pole) is stroked over each paralyzed muscle from ten to twenty times daily. If a permanent contraction take place in paralyzed muscles—*contracture*—an endeavor should be made by *massage* to prevent increase in the condition, and to render it more and more retrogressive. Also, the *galvanic current* by stabile (immovable) application is capable of overcoming muscular contractures. At times **tenotomy** may be serviceable. Certain kinds of paralysis will require **orthopedic apparatus** to neutralize more or less completely the loss of functional activity.

MOTOR PARALYSIS OF THE TRIGEMINAL NERVE (PARALYSIS OF THE THIRD DIVISION OF THE TRIGEMINAL NERVE).

The third or inframaxillary division of the trigeminal nerve contains motor nerve-fibers for the muscles of mastication (masseter, temporal, external and internal pterygoid). If these nerve-tracts are paralyzed, there will result motor or *masticatory trigeminal paralysis* or *paralysis of the muscles of mastication.* The condition is rare, as the deep situation of the third division of the trigeminal nerve affords protection from cold and external injury. *Pressure-paralysis* occurs with relative frequency in consequence of syphilitic or chronic inflammatory thickening of the meninges, particularly of the dura mater at the base of the brain, or of aneurysms of the cerebral arteries, or of syphilitic or tuberculous processes in the sphenoid bone, if these cause constriction of the oval foramen, through which the third division of the trigeminal nerve leaves the skull. Intracranial lesions usually involve the sensory as well as the motor branches of the trigeminal nerve, so that in addition to paralysis of the muscles of mastication there will also be trigeminal anesthesia.

In cases of *unilateral paralysis of the muscles of mastication* it will at once be noted that if the patient be requested to bring the teeth firmly together, the prominence and hardening of the contracted temporal and masseter muscles can be seen and felt only upon the healthy side, while these are wanting upon the diseased side. With each movement of mastication the lower jaw is deflected toward the paralyzed side, and if the patient be requested to move the lower jaw laterally he will be capable of doing this only toward the paralyzed side, but not toward the healthy side, as the latter movement must be performed by the paralyzed pterygoid muscles. When paralysis of the muscles of mastication has existed for some time the temporal and masseter muscles may undergo wasting, and more or less deep excavations form upon the diseased side in the temporal region and in the cheek.

Bilateral paralysis of the muscles of mastication is attended with drooping of the lower jaw and inability to bring the teeth together in mastication or to move the jaw laterally.

PARALYSIS OF THE FACIAL NERVE.

Etiology.—Peripheral paralysis of the seventh cerebral or facial nerve is one of the most common varieties of paralysis, because, by reason of its superficial and comparatively long course, the nerve is readily exposed to refrigeratory and traumatic influences. *Toxic paralysis of the facial nerve,* as, for instance, from lead-poisoning, as well as *infectious paralysis* (following diphtheria, erysipelas, typhoid fever, syphilis, herpes zoster), is rare. *Refrigeratory (rheumatic) paralysis of the facial nerve* occurs with particular frequency when one side of the face is exposed to cold air, especially while the body is over-heated. *Traumatic paralysis of the facial nerve* occurs much more commonly. Even the newborn may be attacked by peripheral paralysis of the facial nerve, if delivery has been effected with **forceps,** and one blade of the instrument, by reason of improper application or of slipping, has exerted undue compression of the trunk of the nerve at the ear or at its division on the cheek. Paralysis of the facial nerve is often associated with **disease of the ear.** Tuberculous destruction of the petrous bone in particular frequently extends to the Fallopian canal and the contained trunk of the facial nerve, causing destruction of the latter. Occasionally the facial nerve is compressed or destroyed and paralyzed by **enlargement or suppuration of the cervical or submaxillary lymphatic glands or of the parotid gland. Incised and contused wounds, operations upon the cheek,** and **severe blows upon the ear** are capable of causing paralysis of the facial nerve. At times peripheral paralysis of the facial nerve is dependent upon **pressure at the base of the skull,** and it may be due to thickening of the dura mater of syphilitic or chronic inflammatory origin, or new-growths or aneurysms. Also, fractures of the skull may be followed by peripheral paralysis of the facial nerve, if the nerve be compressed by hemorrhage or ruptured in fracture of the petrous portion of the temporal bone. Members of nervous families and drunkards appear predisposed to paralysis of the facial nerve in greater degree than others, probably because the resistance of the nervous tissues to injurious influences of various kind has been diminished. Occasionally persons are attacked with peripheral paralysis of the facial nerve a number of times in the course of a few years, at times always upon the same side, and at other times upon one or the other side.

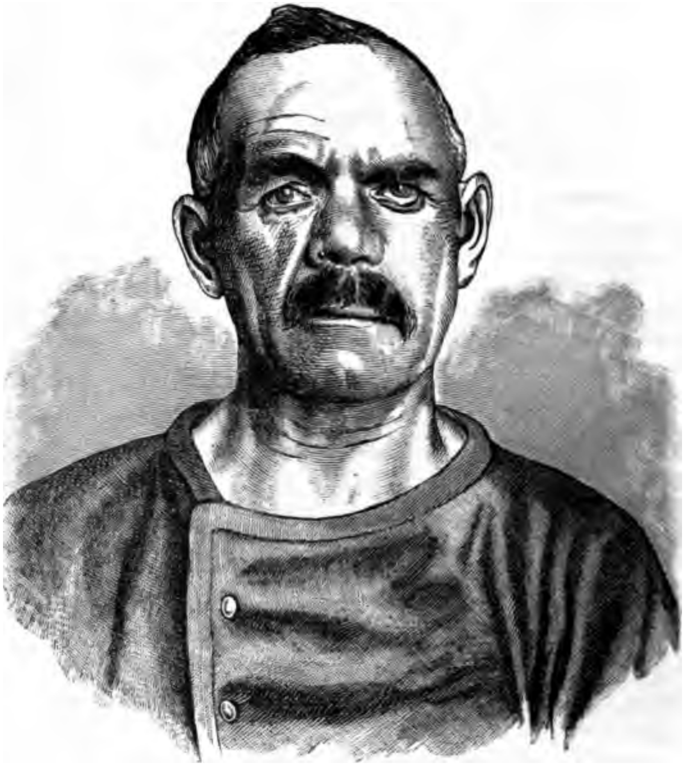


FIG. 60.—Facial expression of a man, 55 years old, presenting severe left-sided traumatic peripheral paralysis of the facial nerve; from a photograph (personal observation, Zurich clinic).

Symptoms.—Of all of the symptoms of peripheral paralysis of the facial nerve, paralysis of the muscles of the face is found unexceptionally. The facial nerve, however, sends motor filaments also to the muscles of the palate and to the stapedius muscle, and accordingly paralysis of this nerve may be attended with

distortion of the uvula and the **arch of the palate**, and **derangement of hearing**. It should not be forgotten that the facial nerve receives from the trigeminal nerve through the greater superficial petrosal nerve gustatory fibers, which leave the trunk of the facial nerve through the chorda tympani; so that **disorders of taste** are possible in connection with facial palsy. Finally, the facial nerve contains secretory fibers for the lacrimal and the salivary glands, and accordingly **derangement in the secretion of tears and of saliva** is at times observed in connection with facial palsy. While paralysis of the muscles of the face is a constant symptom of paralysis of the facial nerve, the occurrence of the remaining symptoms depends upon the situation at which the trunk of the nerve is diseased. Even during rest the paralyzed side of the face is conspicuous for its smooth, flattened appearance, the absence of folds, the greater size of the palpebral fissure than upon the healthy side, the eversion of the lower eyelid, the fulness of the conjunctival sac with tears, and often the flow of tears over the

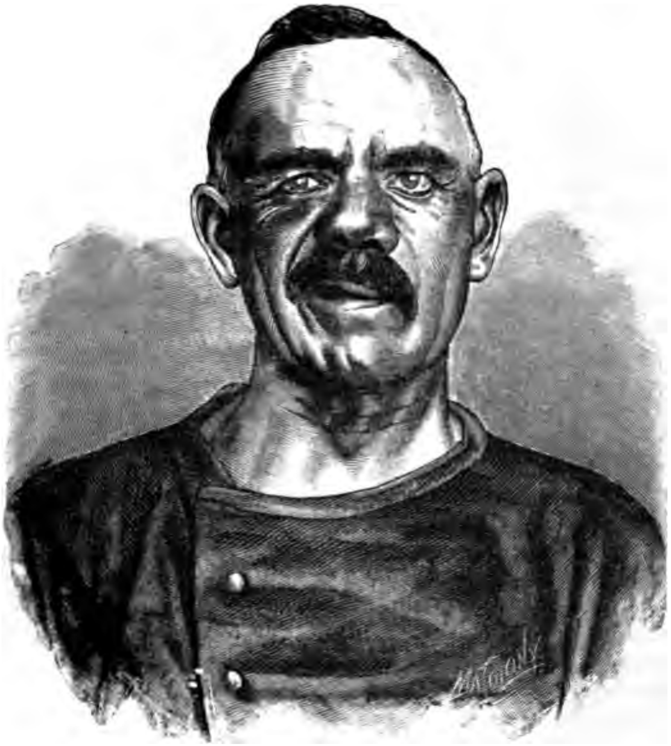


FIG. 61.—The same patient shown in Fig. 60, on laughing.

lower lid across the cheek—so-called epiphora. The attention of the observer is particularly attracted by the unusual phenomenon that closure of the eyelids takes place only upon the healthy side, while the eye upon the diseased side remains constantly open (Fig. 60). The paralyzed side of the face appears drawn toward the healthy side. The difference between the paralyzed and the healthy side of the face becomes the more marked during laughter or on crying, for the paralyzed side remains immobile, inanimate, and mask-like, while the healthy side undergoes natural furrowing and alterations (Fig. 61).

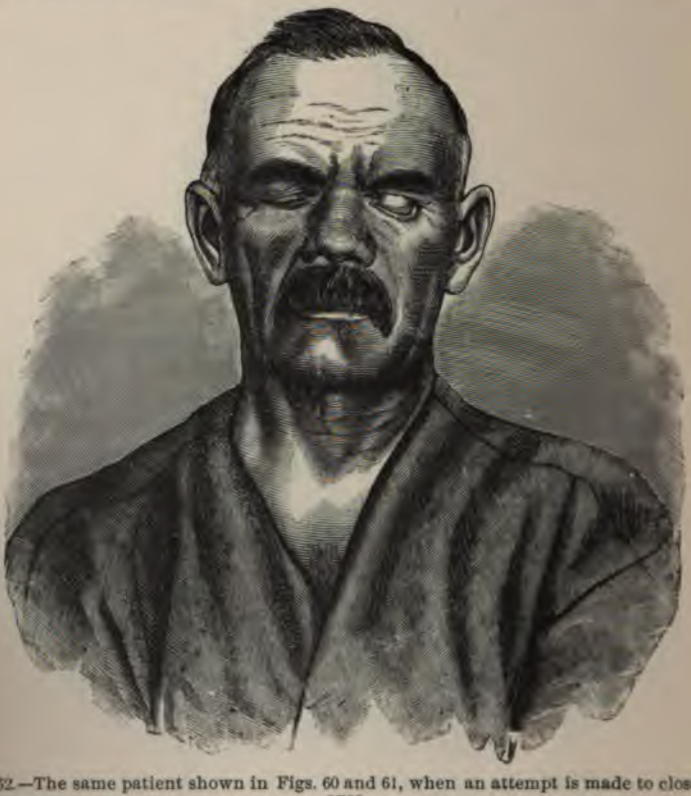


FIG 62.—The same patient shown in Figs. 60 and 61, when an attempt is made to close the eyes.

In order to study more carefully the paralysis of individual muscles, it is well to request the patient to perform given movements with the muscles of the face. It will at once be observed that the **forehead**, even during rest, is free from furrows, as compared with the healthy side. If the patient be requested to wrinkle the forehead, horizontal furrows appear only upon the healthy side, ending at the middle line, while, in consequence of paralysis of the frontal muscle, no furrows are formed upon the diseased side. *Paralysis of the corrugator muscle of the eyebrow* is indicated by the fact that the brow upon the paralyzed side is lower than that upon the healthy side, and that on wrinkling the forehead vertical furrows become apparent only over the glabella upon the healthy side. The greater width of the palpebral fissure upon the diseased side is due to *paralysis of the orbicular muscle of the eyelids.* The action of the elevator muscle of the eyelid, innervated by the oculomotor, then preponderates, drawing the upper lid upward, so that in comparison with the healthy side the fissure appears reduced in size. To the same circumstance is due the eversion of the lower eyelid—paralytic ectropion. Paralysis of the muscle of Horner, a portion of the orbicular muscle toward the nasal aspect, is attended with displacement of the lacrimal caruncle, so that the tears do not properly enter it and the duct continuous with it. This circumstance explains the fact that the eye upon the paralyzed side is constantly bathed in an excess of tears, and that frequently tears flow over the lower eyelid and upon the cheek—so-called **epiphora.** In consequence, the nasal mucous membrane remains dry, and the patients complain of dryness and burning in the nose upon the paralyzed side, and also of impairment of the sense of smell.

Marked disfigurement of the face results in consequence of absence of winking upon the paralyzed side, and even if the finger be applied to the eyeball or the cornea is touched reflex closure of the eye fails to take place. The disfigurement becomes particularly marked if the patient, when requested, attempts to close the eyes, only the eye upon the healthy side being closed, while that upon the diseased side remains open—**paralytic lagophthalmos**—and between the lids the white sclera and generally also a small portion of the cornea become visible as the eyeball is rolled upward and inward on closure of the eyes (Fig. 62).

Frequently, on attempting to close the eyes, a *narrowing of the palpebral fissure* upon the paralyzed side is observed that has been attributed to synergistic relaxation of the elevator of the lids.

The tip of the nose is deflected toward the healthy side, while upon the diseased side the entrance to the nares appears reduced in size. On deep inspiration the ala of the nose upon the diseased side is drawn toward the septum. Persons capable of moving the nasal alæ voluntarily are able in the presence of facial paralysis to make such movement only upon the healthy side. Also, the **buccal orifice** and the **chin** are drawn toward the healthy side. The angle of the mouth is lower upon the paralyzed than upon the healthy side, in consequence of paralysis of the elevator muscle of the angle of the mouth. Whistling and distention of the cheeks th air are impossible, because the lips cannot be closed upon the ralyzed side, and the air is thus permitted to escape from the outh. Also in speaking, and particularly in the use of the labials , p, f, v, w), disturbances and secondary sounds are appreciable, d also in the articulation of certain vocal sounds (a, e). In eat- g and drinking, the ingesta readily escape between the paralyzed d separated lips, so that some patients are compelled to perform ese acts with the head thrown backward.

In consequence of paralysis of the buccinator muscle the **act mastication** is interfered with. The cheeks are not rendered fficiently tense, and as a result the mucous membrane of the eek is readily caught between the teeth in the act of chewing, d is bitten. Also, food readily finds its way between the ucous membrane of the cheek and the teeth, and must be re- oved with the fingers or by pressure upon the cheek from with- t. At times the **base of the tongue** appears lower upon the dis- sed than upon the healthy side in consequence of paralysis of e stylohyoid and digastric muscles. If the muscles of the soft late are involved in paralysis of the facial nerve, the tip of the ula is deflected toward the healthy side, and the arch of the late not only assumes a lower level, as compared with the althy side, but on active respiration flaps like a loose sail.

Paralysis of the musculature of the cheek is revealed not only, has been mentioned, by disturbances in mastication, but also by literation or depression of the **nasolabial fold.** In animated nversation, with respiratory movement, and during sleep the ralyzed cheek flaps to and fro like a curtain.

Paralysis of the muscles of the auricle and of the occipital uscle is frequently demonstrable only with difficulty, because t many persons are capable of moving the auricle voluntarily. aturally, the movements of the auricle would be wanting upon e paralyzed side. Examination of the muscles by means of the radic and the galvanic current could yet be practised, although terations in electric muscular irritability would be expected only the presence of moderately severe paralysis. Involvement of e *stapedius muscle* in paralysis of the facial nerve will be indi- ted by **excessive acuity of hearing—oxyokoia (Willisian hyper- usis)**—which is explained by the preponderant action of the nsor tympani muscle.

Paralysis of the platysma myoides can be recognized from the ct that on marked depression of the lower lip contraction of the atysma on the paralyzed side, with the formation of folds in the in of the neck, does not occur.

It is noteworthy that peripheral facial palsy is attended with sence of all **reflex** and **associated movements.** Movement of the igers toward the eye or contact with the cornea fails to induce osure of the lids, and in crying, laughing, and yawning the paralyzed side remains immobile and inanimate. **Disturbances of taste** are only to be expected in association with peripheral facial palsy when the lesion is situated between the geniculate ganglion and the origin of the chorda tympani. They involve always only the anterior two-thirds of the tongue, as the posterior third is supplied with gustatory fibers by the glossopharyngeal nerve. Often the patient complains of **diminished tactile sensibility of the tongue** on the paralyzed side.

Examination of the *sense of taste* is made most simply by dipping rolls of bibulous paper successively in solutions of common salt, vinegar, sugar, and quinin, and touching the anterior two-thirds of the tongue upon one and then upon the other side. The patient should retract the tongue into the mouth only after he has appreciated a distinct gustatory sensation. Bitter solutions are tested last, because they leave an after-taste for a long time. It is advisable to test the sense of taste also by means of the galvanic current and a gustatory electrode.

Some patients complain of **dryness of the mouth upon the paralyzed side,** and this condition is associated with disorders in the secretory branches of the facial nerve to the salivary glands. If the *nerve-fibers for the lacrimal gland* upon the paralyzed side are also involved, secretion of tears takes place on crying only upon the healthy side. *Sensory disturbances* are wanting in cases of pure facial palsy. Nevertheless, cutaneous anesthesia is now and then observed upon the paralyzed side, and particularly if, as a result of the action of peripheral, injurious influences, in addition to the fibers of the facial nerve, also the terminal fibers of the intimately related trigeminal nerve are involved.

The occurrence of facial palsy is at times preceded by **prodromes,** such as vertigo, headache, mental confusion, ringing in the ears, impairment of hearing, and pain in the ear. At other times the paralysis follows immediately upon the action of the injurious influence (cold, injury). Often the attention of the patient is directed by his friends to the distortion of the face and the paralysis of the facial nerve. In other instances he is induced by a sense of tension, impaired motility, difficulty in eating, speaking, and whistling, to resort to the use of a mirror, when the alteration upon one side of the face is noticed. At times the disorder is discovered quite accidentally on using the mirror in the performance of the toilet. Whether the paralysis is of mild degree, of moderate severity, or profound can be determined alone by *electric examination* (p. 473). Should there be no electric apparatus at hand, the *mechanical irritability* of the paralyzed muscles should be tested, for increased mechanical muscular irritability and tardy muscular contractions are indicative of a moderately severe or a profound paralysis. After the paralysis of the facial muscles has existed for some time *muscular wasting* gradually ensues—*atrophy* due to *inactivity*.

Profound paralysis of the facial nerve may persist throughout the remainder of life, but often only partial improvement ensues, some muscles of the face regaining their motility, while others, on the other hand, remain paralyzed permanently. Persistent muscular contraction may readily develop in paralyzed muscles—*muscular contracture*—and the previously smooth and unfurrowed paralyzed side of the face now becomes distorted by unusually deep furrows. *Involuntary contractions* also occur frequently in the paralyzed muscles, and these may also be induced voluntarily by slight cutaneous irritation. Often an abnormally increased tendency to *associated movements* is noticeable. On closure of the eyes, for instance, twitching occurs at the angle of the mouth, or, on puckering the lips, contraction of the palpebral fissure. Long-continued paralysis of the facial nerve may be followed by *disease of the eye*, for in the absence of winking, and if the cornea is not moistened with tears and cleared of foreign bodies from the air, there will be danger of inflammation of the cornea (keratitis), rupture of the cornea, and loss of the eyeball (panophthalmitis).

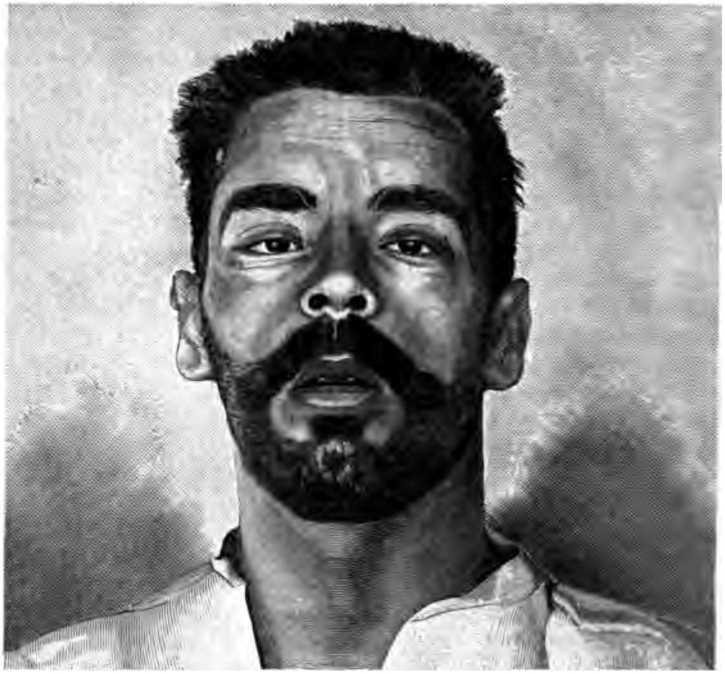


FIG. 63.—Facial expression in a case of bilateral facial paralysis; from a photograph (personal observation, Zurich clinic).

Among rare *complications* of peripheral facial palsy, *vasomotor and trophic disturbances* appear at times upon the paralyzed side, such as pallor of the

grayness of the hair, labial, lingual, and pharyngeal herpes. The
es may precede the paralysis or follow it after the lapse of a few days.
mes *hyperidrosis* appears upon the paralyzed side.

'eripheral paralysis of the facial nerve is generally unilateral
cial hemiplegia; bilateral paralysis of the facial nerve—*facial*
gia—is encountered but rarely. Either the latter is the
lt of tuberculous disease of both petrous bones, paralysis in-
ing at first the one and then the other facial nerve; or it is
iated with lesions at the base of the skull, and here at times
ltaneously if the basal disease involves both facial nerves at

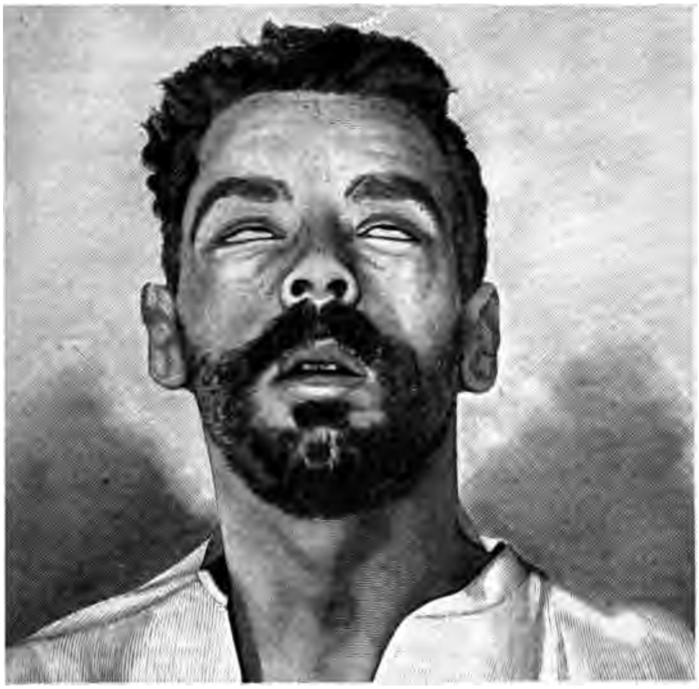


Fig. 64.—The same patient as in Fig. 63, on attempting to close the eyes.

same time; or both nerves may be affected in quick succession
old or external injuries. Further, examples are known also
hich the facial nerve upon one side was paralyzed as a result
eripheral influences, and that upon the opposite side as a result
entral influences, the arm and the leg being involved addi-
lly in the paralysis in association with the latter, with the
lopment of the clinical picture of cerebral hemiplegia. In
presence of *facial diplegia* the face has lost its power of ex-
sion upon both sides, remains inanimate, and resembles a
k. The patients may be heard to laugh or shout or cry, but

the facial expression remains rigid and unaltered. The upper lip not rarely droops like a proboscis, while the lower lip is everted. The imperfect closure of the lips prevents in marked degree the ingestion of nutriment, because food and drink readily escape from the mouth. Often the patients introduce the food deeply into the pharynx with the fingers, a spoon, or a specially devised wooden spatula, in order to facilitate swallowing. When the patient is requested to close the eyes both palpebral fissures persist (Figs. 63 and 64).

Anatomic Alterations.—Little is known with regard to the anatomic alterations of peripheral facial paralysis, because the disorder is not fatal, and because if death should result accidentally from other causes the removal of the nerve and the facial muscles will not be permitted on account of the unavoidable mutilation of the face. In severe cases of rheumatic or traumatic paralysis of the facial nerve destruction, fatty degeneration, and disappearance of the medullary sheaths and axis-cylinders, and multiplication of the nuclei of the sheaths of Schwann, have been found in the peripheral portions of divided nerves. The connective tissue of the nerves has generally been unaltered. Fatty degeneration had taken place in the paralyzed facial muscles.

Mild facial paralysis is perhaps attended with scarcely more than active over-distention of the vessels with blood, and slight exudation into the connective-tissue nerve-sheath, with compression of the nerve-fibers, without extensive nerve-degeneration. Therefore, these conditions are susceptible of speedy involution.

Diagnosis.—The recognition of peripheral facial palsy is easy on account of the striking distortion of the face. In contradistinction from **central facial paralysis,** all of the facial muscles are involved, while with central facial paralysis the ***branch to the forehead*** remains uninvolved, so that the patient is capable of wrinkling the forehead and eyebrows, and of closing the eyes. In addition, with central paralysis reflex and associated movements are preserved; phenomena of degenerative electric reaction are wanting; and, finally, the arm and the leg are, as a rule, involved, so that a condition of hemiplegia exists. Only with disease of the *bones* does facial paralysis preserve its peripheral character if the lesion involves the intrapontine facial path from the point of exit of the facial trunk at the posterior peduncle of the pons to the facial nucleus, or the latter itself. Other pontine symptoms (interference with swallowing, contracted pupils, difficulty in articulation) will then usually be present also, and frequently also paralysis of the extremities.

The *seat of peripheral facial paralysis* can be readily determined with the aid of the accompanying diagram representing the course of the facial nerve (Fig. 65).

1. If the *lesion is situated outside the stylomastoid foramen,* the

paralysis will be confined to the *muscles of the face* alone (Fig. 65, 1).

2. A *lesion in the lowermost portion of the Fallopian canal* will be attended in addition with paralysis of the posterior auricular nerve, and consequently of the *muscles of the auricle* and the *occipital muscle* (Fig. 65, 2).

3. When the *lesion is situated above the point of origin of the chorda tympani nerve* there will be, in addition to paralysis of the muscles of the face and of the auricle, also disturbances in the sense of taste and of the secretion of saliva (Fig. 65, 3).

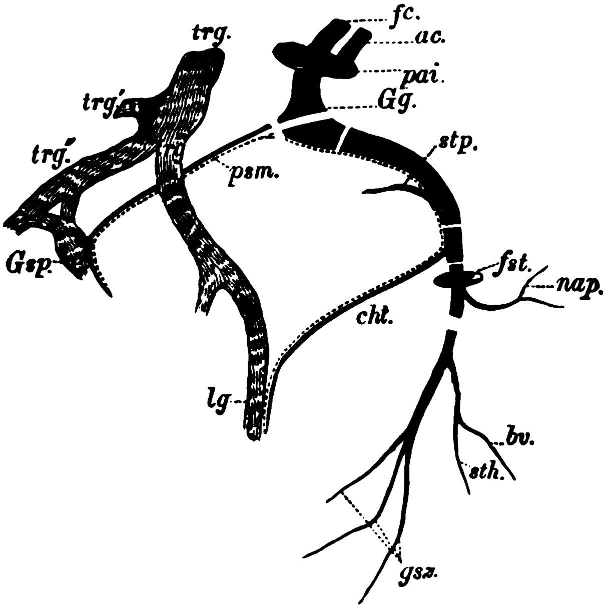


Fig. 65.—Diagram of the distribution of the facial nerve: *fc*, trunk of the facial nerve; *ac*, trunk of the auditory nerve; *pai*, internal auditory canal; *Gg*, geniculate ganglion; *psm*, greater superficial petrosal nerve; *stp*, stapedius nerve; *cht*, chorda tympani; *fst*, styloid foramen; *nap*, posterior auricular nerve; *bv* and *sth*, nerves for the digastric and stylohyoid muscles; *gsz*, facial branches; *trg*, *trg'*, *trg''*, *trg'''*, trunk and three branches of the trigeminal nerve; *Gsp*, sphenopalatine ganglion; *lg*, lingual nerve.

4. If the facial nerve is diseased *between the point of origin of the stapedius nerve and the geniculate ganglion, disorders of hearing* (*oxyokoia*) will be observed, in addition to paralysis of the muscles of the face, the auricle, and the occipital muscle, and to disturbances in the sense of taste and in the secretion of saliva (Fig. 65, 4).

5. *Disease of the geniculate ganglion* itself will be attended, in addition to paralysis of the facial muscles, the muscles of the auricle, the occipital muscle, disturbances in the sense of taste, in

the secretion of saliva, and in the sense of hearing, also with *paralysis of the uvula* and *suppression of the secretion of tears*, as the fibers of the facial nerve governing the functions last named leave the path of the nerve at the geniculate ganglion, in order to join the path of the trigeminus through the greater superficial petrosal nerve (Fig. 65, 5).

6. *Lesions above the geniculate ganglion* will be attended with all of the manifestations of facial palsy except *disturbances in the sense of taste.* There will thus be present paralysis of the uvula, derangement in the secretion of tears, in the sense of hearing, in the secretion of saliva, paralysis of the facial muscles and those of the auricle, and the occipital muscle.

The **prognosis** and the **treatment** will be governed by the rules laid down on pp. 475 and 476.

PARALYSIS OF THE SPINAL ACCESSORY NERVE.

In the discussion of peripheral paralysis of the spinal accessory nerve the motor accessory fibers that enter the vagus, and supply especially the muscles of the larynx and the pharynx, and pass to the cardiac plexus, are omitted from consideration. The condition to be described is attended only with *paralysis of the sternomastoid and the trapezius.* Most commonly, paralysis of the spinal accessory nerve is of *traumatic* origin, as this nerve pursues a long and superficial course upon either side of the neck. Stab-wounds, operation-wounds, suppurating cervical lymphatic glands, destroying the trunk of the spinal accessory nerve, are relatively frequent causes of such paralysis. Nevertheless, paralysis of the spinal accessory nerve is one of the less common varieties of peripheral paralysis. At times paralysis of the spinal accessory is dependent upon disease of the cervical vertebræ, as it sends numerous root-filaments to the level of the seventh cervical vertebra.

In the presence of *unilateral paralysis of the sternocleidomastoid muscle* the head and the chin acquire an abnormal position, in consequence of predominant activity of the healthy sternomastoid. *The head and the chin are directed toward the paralyzed side, and the chin is elevated.* Rotation of the head in the opposite direction is greatly interfered with. When the patient lies upon his back and attempts to raise his head the contracted sternomastoid becomes apparent beneath the skin only upon the healthy side of the neck. Also, on deep inspiration only the healthy and not the paralyzed sternomastoid undergoes contraction. If the paralysis has existed for some time, a depression in the course of the neck is observed upon the paralyzed side, resulting from inactivity-atrophy of the paralyzed sternomastoid. At times the healthy sternomastoid undergoes contracture, with the development of [illegible] which greatly prevents rotatory movement of the head. In the presence of *bilateral paralysis of the sternomastoid* the patient is almost wholly incapable of raising his head from the recumbent posture.

Paralysis of the trapezius muscle is indicated, on inspection of the patient from the front, by the fact that the *supraclavicular fossa* upon the paralyzed side is unusually deep, and the shoulder is lower. Viewed from the posterior aspect the *scapula* upon the paralyzed side is lower than upon the healthy side. At the same time, the scapula upon the paralyzed side stands off a greater distance from the vertebral column, its upper and outer angle being drawn markedly forward and downward by the weight of the arm, while the inferior angle is more closely approximated to the vertebral column. *Movement of the scapula* is rendered difficult, as it can be accomplished only by contraction of the elevator of the angle of the scapula. The act of *approximating the scapula to the vertebral column* is rendered difficult, and is possible only through the agency of the rhomboid. *Elevation of the arm above the horizontal level* is attended with difficulty, because the scapula is imperfectly fixed. Further, the external free border of the paralyzed trapezius does not form a straight line, but a curved line, with its convexity directed toward the vertebral column. In the presence of *bilateral paralysis of the trapezius* the back appears unusually wide. In addition, the conditions just described are present on both sides.

PARALYSIS OF THE HYPOGLOSSAL NERVE.

Peripheral paralysis of the hypoglossal nerve gives rise to *paralysis of the tongue—glossoplegia*. The paralysis of the tongue in turn is characterized in part by disorders of mastication and in part by disorders of speech, and accordingly it may be divided into *masticatory* and *articulatory glossoplegia*. Hypoglossal palsy is most commonly of *traumatic origin*, resulting especially from punctured and incised wounds, or from pressure exerted by enlarged lymphatic glands or other tumors of the neck. I have recently treated a woman with hypoglossal paralysis in the sequence of syphilis, induced by gummatous hyperplasia upon the dura at the base of the skull. After a course of treatment with inunctions of mercurial ointment and the internal administration of potassium iodid complete recovery ensued.

Unilateral hypoglossal paralysis is attended with *deviation of the tip of the tongue toward the paralyzed side* in consequence of preponderant action of the healthy genioglossus muscle. The dorsum of the tongue is the more greatly arched upon the paralyzed side. The diseased half of the tongue exhibits *more marked furrowing of its surface*, and often also active fibrillary twitching. In *chewing*, the food readily remains upon the paralyzed half of the tongue, and the patients often complain in consequence of imperfect distribution of the food in the mouth and of defective taste. In the act of deglutition deficient propulsion of the food is appreciable, and the approximation of the tongue to the palate is incomplete, so that food may readily return into the mouth. The swallowing of saliva may also be interfered with, and the patient in consequence often permits dribbling from the mouth. In *speaking*, the imperfect articulation, particularly of the lingual letters (d, t, s, sch, l, r, k, g, ch), will be noted. If the paralysis has existed for some time, *unilateral atrophy of the tongue* takes place.

In cases of *bilateral hypoglossal paralysis* the tongue is converted into an immobile muscular mass. The formation of a bolus is scarcely possible, and speech is reduced to an inarticulate grunt or jargon, so that the patient can often make himself understood only in writing.

MULTIPLE OR COMBINED PARALYSIS OF CEREBRAL NERVES.

In the presence of multiple or combined paralysis of cerebral nerves several cerebral nerves may be paralyzed together. At times almost all of the cerebral nerves upon one side are paralyzed, while in other instances some nerves may be paralyzed upon one side and others upon the opposite side of the body. Such a condition is most commonly associated with lesions at the base of the skull, which extend gradually and involve one nerve after the other. Particularly, *syphilitic, carcinomatous, and tuberculous lesions of the meninges and the cranial bones* are the etiologic factors. Less commonly, *aneurysms of the cerebral arteries* or *fracture of the base of the skull* is the cause of the disorder. At times multiple paralysis of cerebral nerves occurs in the sequence of *infectious diseases*, as, for instance, after pharyngeal diphtheria. It occurs occasionally, also, in the course of *polyneuritis*. *Injuries in the upper portion of the cervical region* are likewise capable of causing paralysis simultaneously of several cerebral nerves. In the differential diagnosis paralysis due to lesions of the nuclei of the cerebral nerves in the medulla oblongata and in the pons is particularly to be taken into consideration. The etiology and the mode of development of the palsy afford the principal means of distinction in the differential diagnosis.

PARALYSIS OF THE PHRENIC NERVE.

The phrenic nerve, which receives fibers from the third and fourth cervical nerves, is paralyzed at times in consequence of *injuries*, as, for instance, fractures, luxations, neoplasms of the cervical vertebræ, tumors, abscesses, punctured and incised wounds of the neck. *Infectious paralysis* of the phrenic nerve has been observed also in the sequence of diphtheria and influenza. *Toxic paralysis* has been noted in the sequence of poisoning with lead, alcohol, carbon monoxid, and opium. At times *refrigeratory paralysis* is observed. It is noteworthy that paralysis of the diaphragm is much more commonly of myopathic than of neuropathic origin; that is, dependent not upon disease of the phrenic nerve, but upon disease of the diaphragm itself. Such a condition occurs particularly in association with diaphragmatic pleurisy and peritonitis, because the inflammation of the serous membrane readily extends to the muscular structure of the diaphragm, whose function is thereby deranged. Paralysis of the diaphragm may involve a number of circumscribed areas, or be unilateral or bilateral.

Bilateral or complete paralysis of the diaphragm is a most dangerous disorder, as death may result from *asphyxia*. During quiet breathing symptoms may naturally be absent, but these appear at once upon active bodily exercise, as, for instance, in ascending stairs. It is noteworthy that the *type of respiration* is purely costal, and that principally the upper portion of the thorax moves in the act of breathing, while the lower remains immobile and appears unusually full. In contrast with normal conditions *inspiratory retraction and expiratory protrusion of the epigastrium* take place. The *upper border of the liver* is unusually high, and scarcely participates in the respiratory movements. The latter, however, are reversed as compared with the normal. The liver ascends with inspiration, as does also its lower border, whose movement frequently can be followed with the finger. All *expulsive efforts* (coughing, defecation, heavy lifting, vomiting, loud and continued speaking or singing) are difficult and incomplete. Even slight catarrhal states of the air-passages may at times be attended with danger of asphyxia, because in consequence of deficient cough and expectoration accumulation of secretion in the air-passages readily takes place. The greater the embarrassment of respiration the more rapid becomes the act of breathing and the greater the cyanosis.

To overcome the dangers of paralysis of the diaphragm *electric treatment* particularly is to be advised. The phrenic nerve will be found in the angle formed between the sternomastoid and the omohyoid muscles. In this situation upon either side an electrode is placed, and a strong faradic current is passed through the diseased nerve, with interruption by means of a suitable device corresponding to the rhythm of normal breathing. Contraction of the diaphragm will be recognized from the fact that air enters the air-passages with a gurgling sound and the epigastrium bulges with inspiration.

PARALYSIS OF THE RADIAL NERVE.

Etiology.—As the facial is the most commonly paralyzed of the cerebral nerves, so among the nerves of the arm the radial is by far the most commonly affected by peripheral paralysis. Its long and superficial course affords a favorable point of attack for external injurious influences. Only rarely is radial paralysis due to **refrigeratory** or **infectious** causes. *Radial paralysis of toxic origin* is distinctly more common. For a long time it has been known particularly that under the influence of lead (saturnism) radial paralysis occurs not rarely. This saturnine paralysis of the radial nerve is characterized by its **bilateral distribution.** Toxic radial paralysis may, however, occur also as a result of the action of alcohol. It has often been observed after injections of ether, antypyrin, and osmic acid beneath the skin of the forearm. *Traumatic radial paralysis* is the most common variety. *Sleep-paralysis* is the best known form, occurring during sleep when the patient places the forearm (usually the right) in such a position that the radial nerve is compressed, and as a result is paralyzed. At times the patient has supported himself upon his arm, resting the upper arm upon a sharp edge, or permitting the arm to hang over a sharp edge (arm of a chair or edge of the bed), or the patient has slept with the arm beneath the head, the upper arm being thereby unduly compressed upon its dorsal aspect, or the lateral decubitus is assumed in sleep, and the radial nerve of the arm placed beneath the body is injured by pressure. The deeper the sleep the less will the inconvenience of a given posture be appreciated, and the greater, therefore, is the danger of pressure-paralysis. Laborers who take a midday nap in their work-places, and drunkards, are therefore attacked by sleep-paralysis with particular frequency. Another important variety of traumatic paralysis of the radial nerve is known as *crutch-palsy.* This develops at times even within a few hours after the use of an unsuitable crutch, particularly if this be too short or too long, or unpadded, and is supplied with unsuitable supports for the hands. Of all the nerves of the arm, the radial in particular is peculiarly exposed to the danger of compression by a crutch on account of its posterior situation in the axilla.

A few other *varieties of traumatic radial paralysis* may be mentioned. Radial paralysis after shackling of the upper arm has been designated *prisoners' palsy.* *Coachmen's palsy* has been observed in a number of instances in Russian coachmen who have wrapped the horses' reins too firmly about the upper arms, and have then, usually in a drunken state, fallen asleep. A *bandage-palsy* also has been described by Russian physicians, occurring in infants when the arms are too firmly bandaged to the trunk, and the infants are, besides, permitted to lie upon one side of the body for too long a time. *Water-carriers' palsy* of Rennes arises from the carrying of heavy water-jugs, through the handles of which the upper arms are passed, the vessels being compressed against the abdomen. In the same way the carrying of heavy bundles by means of straps or ropes wound about the upper arms may give rise to radial paralysis. Under some conditions the *application of Esmarch's rubber tube to the arm* is followed by peripheral pressure-paralysis of the radial nerve, and an improperly applied *plaster-of-Paris bandage* may also have the same effect. The designation *narcosis-paralysis* has been applied to cases of radial palsy that have developed during narcosis, in the course of an operation, in consequence of compression of the radial nerve by the head of the humerus in lifting the arm upward. Naturally, *punctured, incised, contused*, and *gunshot* wounds may cause injury and paralysis of the radial nerve. Occasionally radial paralysis develops in the sequence of *luxation* or *fracture of the humerus*. In the latter event the radial nerve may be lacerated and paralyzed by the extremities of the fractured bone, or it may be surrounded by callus and be paralyzed by compression. Examples of traumatic radial paralysis could be largely multiplied. *Drummers' paralysis* may yet be mentioned, involving most commonly the long extensor of the thumb and resulting from over-use of the muscles in beating the drum.

Anatomic Alterations.—Little is known with regard to the anatomic alterations attending peripheral radial paralysis, because the disorder in no sense is dangerous to life. In a case of radial paralysis following typhus fever neuritic alterations were found, and in a case of recent lead-palsy I succeeded in finding throughout the entire nervous system only degenerative atrophy of the fibers of the radial nerve.

Symptoms and Diagnosis.—Individuals with radial paralysis attract attention at once by the **position of the hand and the fingers.** When the arm is raised vertically the hand is flexed upon the palm, and at the same time pronated. The thumb is adducted and slightly flexed, and the remaining fingers likewise are flexed upon the palm (Fig. 66). If the patient be directed to practise dorsal flexion with the paralyzed hand, this will be found impossible on account of paralysis of the radial and ulnar extensors of the carpus. As a result of paralysis of the common extensor of the fingers the patients are unable to perform dorsal flexion of the basal phalanx of the fingers. Extension of the fingers is not

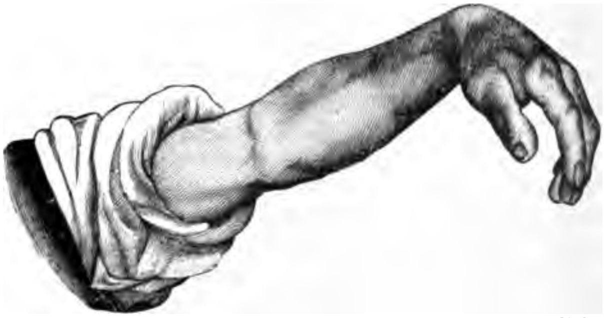


FIG. 66.—Position of the fingers and the hand in a case of peripheral radial paralysis—sleep-paralysis; from a photograph (personal observation, Zurich clinic).

at all possible, as extension of the second and third phalanges, which are supplied by the interosseous muscles (with synchronous flexion of the basal phalanges), can take place only if the basal phalanx has been previously flexed upon the dorsum. Extension of the fingers can, however, be effected when the basal phalanges have been passively placed in dorsal flexion. Extension of the thumb cannot be effected because the long and short extensors of this digit are paralyzed; abduction also is greatly limited in consequence of paralysis of the long abductor of the thumb. The abolition of movement in the fingers and the thumb causes great difficulty in the execution of all fine manipulations (drawing, writing, buttoning). The grasp of the hand is extremely feeble, because, in consequence of paralysis of the extensors of the fingers, the points of attachment for the flexors are not sufficiently distant. If the forearm and the hand are placed upon a horizontal support, the patient will be unable to abduct the hand toward the radial or the ulnar aspect, as the long and short radial extensors of the carpus and the ulnar extensor of the carpus are paralyzed. When, however, the hand is held in the dependent posture these movements can be executed by the corresponding flexors. Often radial paralysis is limited to the muscles already mentioned, as the nerve-filaments for the long and short supinators and the triceps leave the trunk of the nerve quite high up, and are frequently situated above the point of injury. Involvement of the short supinator in radial paralysis is attended with inability to execute supination of the forearm when this member is held in the dependent posture. Paralysis of the long supinator may be recognized by the absence of contraction of the muscle named when resistance is offered to flexion of the forearm held in a position midway between pronation and supination. Paralysis of the triceps, finally, is attended with inability to extend the flexed forearm, or to offer resistance to passive flexion of the forearm.

The **alterations in electric irritability** of the paralyzed nerves and muscles vary in accordance with the severity of the palsy. In the presence of slight and moderately severe paralysis the electric current is a convenient means for determining the seat of the paralysis, for if the course of the radial nerve be followed with the electrode of a faradic current muscular contractions will be wanting above the seat of the lesion, and will be observed only below this point on stimulation of the nerve. After the paralysis has existed for some time *atrophy from want of use* develops in the paralyzed muscles.

Although the radial is a mixed nerve, *sensory disturbances* may be wanting in a case of radial paralysis because the sensory branches are given off from the trunk of the nerve at so high a level that they are generally situated above the lesion, or because neighboring nerves assume the function of the paralyzed radial branches. When sensory disturbances are present these involv the dorsal aspect of the upper arm (posterior superior cutaneou nerve), that of the forearm (posterior inferior cutaneous nerve and that of the hand and the fingers between the thumb and th middle of the middle finger. The terminal phalanges naturall remain unaffected, as these are supplied with cutaneous branche by the median nerve.

Vasomotor and *trophic disorders* occur but exceptionally, as, for instanc nodular thickening of the extensor-tendons or thickening of the digit articulations.

The *onset of paralysis of the radial nerve* is often sudden, th patient awaking from deep sleep therewith. In other instance however, the condition is preceded by *prodromes*—paresthesia, suc as a sense of contraction, of the crawling of ants, of coldness an stiffness, and the like. The *duration of radial paralysis* generall is exceedingly long. Even mild sleep-palsies may persist fro four to six weeks. *Repeated radial paralysis* may occur in laborer and drunkards if compression of the nerve is repeated durin sleep. Plumbic paralysis of the radial nerve likewise may readil recur if the individual is again exposed to the action of lead.

PARALYSIS OF THE MEDIAN NERVE.

Etiology.—Peripheral paralysis of the median nerve is, lik that of the radial nerve, generally of **traumatic origin.** Th median nerve is injured with particular frequency just above th wrist-joint, upon the radial aspect of the palmar surface of th forearm by incised wounds, as, for instance, by fragments of glas while injuries of the trunk of the nerve in the internal bicipita sulcus are much less common. Nevertheless, *crutch-paralysi prisoners' paralysis,* and *narcosis-paralysis* of the median nerv as well as paralysis in consequence of *luxation* and *fracture of th humerus,* have been observed. At times median paralysis has bee observed after **excessive muscular contraction.** In this group i included drummers' paralysis, which involves especially the flexo of the thumb, and naturally much more commonly the long extensor of the thumb supplied by the radial nerve. *Refrigeratory* an *infectious paralysis* of the median nerve is uncommon.

Symptoms and Diagnosis.—The extent of the paralysi naturally depends upon the seat of the lesion. If the media nerve is injured just above the wrist-joint, the muscles of th thenar eminence are paralyzed, with the exception of the adducto of the thumb, supplied by the ulnar nerve, and in consequence o loss of function on the part of the short abductor of the thum the short flexor of the thumb, the opposing muscle of the thum the power of abduction and flexion of the thumb is partially an the opposing action of the thumb is wholly abolished. Whe the paralysis has existed for some time atrophy of the thenar muscles takes place. As the action of the abductor of the thumb, supplied by the ulnar nerve, preponderates, the thumb becomes permanently approximated to the hand, and its terminal phalanx is markedly flexed upon the dorsum through the action of the long and short extensors of the thumb. As a result there develops a form of hand that has been designated the *ape-hand*. There are paralyzed, in addition, the first and second lumbrical muscles, which effect palmar flexion of the second and third phalanges of the index-finger and the middle finger, with synchronous dorsal flexion of the basal phalanx. The **sensibility** of the skin is lost upon the radial aspect of the palmar surface of the hand to a median line passing through the fourth finger. Upon the dorsal aspect of the finger cutaneous sensibility is wanting in the terminal phalanges of the thumb and of the second and third fingers. Not rarely **trophic alterations** occur, particularly glossy finger, vesication of the skin, thickening and fracture of the nails. A lesion in the course of the internal bicipital sulcus causes paralysis of all of the muscles upon the palmar surface of the forearm, in addition to the muscles already named, with the exception of the ulnar flexor of the carpus, which is supplied by the ulnar nerve. The patient is, therefore, unable to flex the terminal phalanx (paralysis of the deep flexor of the digits) and the second phalanx (paralysis of the superficial flexor of the digits). This is particularly noticeable in the second and third fingers, and less so in the fourth and fifth, as the deep flexor receives besides branches from the ulnar nerve. Palmar flexion of the hand can be effected only with difficulty, and with synchronous abduction toward the ulna (from contraction of the ulnar flexor of the carpus). In consequence of paralysis of the radial flexor of the carpus abduction of the hand toward the radius is impossible if the hand is in a

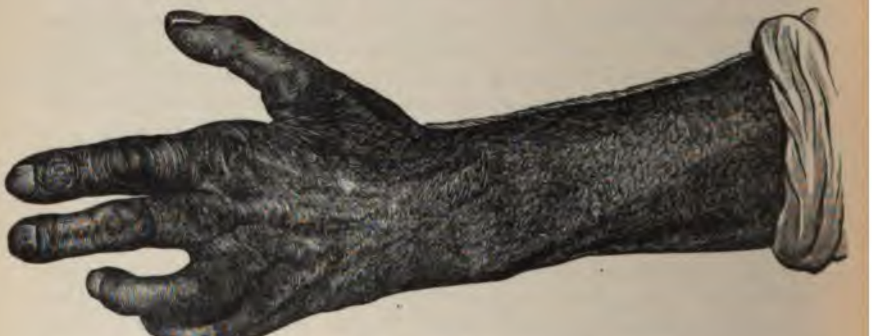


FIG. 67.—Claw-hand following paralysis of the left ulnar nerve as the result of a punctured wound; from a photograph (personal observation, Zurich clinic).

position of palmar flexion. Paralysis of the round pronator and of the quadrate muscle prevents pronation of the dependent arm, while with the forearm flexed the long supinator (supplied by the radial nerve) will effect pronation of the forearm.

PARALYSIS OF THE ULNAR NERVE.

Etiology.—*Refrigeratory and infectious paralysis of the ulnar nerve* is uncommon. The condition is generally dependent upon **traumatic influences**, which coincide with those of traumatic radial paralysis. Sleep-palsy, crutch-palsy, narcosis-palsy, and prisoners' palsy involving the ulnar nerve have been observed. At times pressure-paralysis of the ulnar nerve has been noted in certain artisans (clockmakers, glassworkers) as a result of long-continued support of the arm upon the elbow.

Symptoms and Diagnosis.—Paralysis of the ulnar nerve is attended with paralysis of the muscles of the hypothenar eminence (abductor, flexor, and opposing muscle of the fifth digit), and consequently the little finger is incapable of executing the corresponding movements. Paralysis of those portions of the deep flexor of the digits passing to the ring-finger and the little finger prevents plantar flexion of the terminal phalanx of these fingers. Plantar flexion of the hand is impaired, and can be performed only toward the radial but not toward the ulnar aspect (paralysis of the ulnar flexor of the carpus). The interosseous muscles are paralyzed, and as a consequence the patients are unable to perform plantar flexion of the basal phalanx of the fingers, and at the same time to extend the other two phalanges. In the presence of paralysis of the ulnar nerve the third and fourth lumbrical muscles are further paralyzed. In consequence extension of the basal phalanx with synchronous plantar flexion of the other two phalanges is wanting. **Sensory disturbances** are present on the palmar aspect of the hand toward the ulna from a median line passing through the ring-finger. On the dorsal surface of the hand and the fingers the sensory disturbance extends to a median line passing through the middle finger and upon the ulnar side of this line.

After the paralysis has existed for some time **atrophy of the** paralyzed muscles takes place *from disuse*. This is appreciable particularly in the muscles of the hypothenar eminence, which presents a thin and flattened appearance, and in the interosseous spaces, which present unusually deep depressions. At times a **claw-hand** develops. This results from dorsal hyperextension of the basal phalanges of the fingers by the common extensor of the digits, while in consequence of paralysis of the lumbrical muscles the flexors of the fingers cause such marked palmar flexion of the other two phalanges that at times the nails are dug into the palm of the hand. As the first and third lumbrical muscles are not involved in paralysis of the ulnar nerve, the claw-position is generally more pronounced in the fourth and fifth fingers (Fig. 67).

PARALYSIS OF THE MUSCULOCUTANEOUS NERVE.

Paralysis of the musculocutaneous nerve occurs with extreme rarity. Paralysis of the biceps of the arm is most commonly attended with loss of flexion of the forearm or with incomplete flexion in pronation from the action of the long supinator supplied by the radial nerve.

PARALYSIS OF THE AXILLARY NERVE.

Etiology.—Paralysis of the axillary nerve is most commonly of traumatic origin (fall, blow, contusion of the shoulder, pressure by a crutch, luxation or fracture of the head of the humerus). At times it occurs as a complication of *inflammation of the shoulder-joint,* perhaps from extension of the inflammatory process to the nerve. *Refrigeratory* and *toxic paralysis* of the axillary nerve (lead-poisoning, diabetes mellitus) are also known.

Symptoms and Diagnosis.—Paralysis of the axillary nerve is attended with **paralysis of the deltoid muscle.** Although the axillary nerve sends fibers to the small round muscle, no disturbance of function is observed in this muscle, as it receives nerve-fibers also from the suprascapular nerve. When the *deltoid muscle is paralyzed* the patient is unable to elevate the arm to the horizontal level. The muscle undergoes gradual atrophy, the shoulder becomes flattened and thin, and a **flail-joint** develops at the shoulder, in consequence of the traction of the arm and the deficient resistance of the paralyzed deltoid muscle. **Sensory disturbances** may be present upon the upper, outer, and posterior aspect of the arm.

COMBINED PARALYSIS OF THE NERVES OF THE ARM AND THE BRACHIAL PLEXUS.

By combined paralysis of the nerves of the arm is understood simultaneous paralysis of several nerves of this member. The cause may be situated in the upper arm or the forearm or in the brachial plexus. The last situation is particularly adapted for the development of combined paralysis of the nerves of the arm, because here the paths for the various nerves to the arm lie so closely together that even a small lesion is capable of injuring several nerves at the same time. Constrictions of the arm, fractures, luxations, and the pressure of a crutch are well adapted to injure several nerves of the arm at the same time in their course through the member.

Paralysis of the brachial plexus also is generally of traumatic origin, and occurs particularly in consequence of pressure in association with luxation of the humerus, dislocation or fractures of the clavicle, and neoplasms of the neck. At times it has been observed to set in abruptly, in an *apoplectiform* manner, spontaneously, or after excessive strain. On post-mortem examination hemorrhage into the brachial plexus has been observed in a number of instances. In all cases of *brachial paralysis* motor disturbances invariably preponderate; sensibility may be wholly unaltered. In accordance with the seat of the lesion several varieties of paralysis of the brachial plexus can be distinguished, namely, an upper, a lower, and a mixed variety, the last including the manifestations of the other two.

The upper variety of paralysis of the brachial plexus involves the fifth and sixth cervical nerve-roots. It includes *Erb's paralysis of the brachial plexus*, which is attended with paralysis of the deltoid, the biceps, the internal brachial, and the long supinator, and also at times of the supraspinous, infraspinous, and short supinator. Erb showed that two or three centimeters above the clavicle, at the level of the transverse process of the sixth cervical vertebra, just behind the external border of the sternomastoid muscle there is a point—*Erb's supraclavicular point*—corresponding to the fifth and sixth cervical nerve-roots, electric stimulation of which induces contraction of the muscles involved in Erb's paralysis of the brachial plexus.

Parturitional or *obstetric paralysis* also belongs in this category. This is observed in the newborn when during labor it has been necessary to free the arms or apply the fingers over the clavicles, or introduce a hook or the fingers into the axillary cavity. Obstetric palsy may result also from slipping of the forceps and pressure upon the brachial plexus. Generally, in addition to the paralysis there is found luxation or fracture of the humerus or fracture of the clavicle or of the scapula. The muscles involved are the same as those that suffer in Erb's palsy. Recovery is not impossible.

The lower variety of paralysis of the brachial plexus involves the distribution of the eighth cervical and the first dorsal nerve. The muscles paralyzed include those of the thenar and hypothenar eminences, the interosseous, and at times also some of the flexors of the forearm. *Oculopupillary symptoms* (contraction of the pupil, narrowing of the palpebral fissure, retraction of the eyeball) will be especially conspicuous. These depend upon the circumstance that the communicating branch of the first dorsal nerve transmits oculopupillary fibers from the cervical cord to the path for the cervical sympathetic. *Sensory disturbances* may be observed especially in the distribution of the median and the ulnar nerve and on the inner aspect of the upper arm.

PERIPHERAL PARALYSIS OF THE SCAPULAR MUSCLES.

PARALYSIS OF THE SERRATE MUSCLE.

Etiology.—The greater anterior serrate muscle receives fibers from the long thoracic nerve, a branch of the brachial plexus. Peripheral paralysis of the serrate muscle is not uncommon, as the nerve pursues a superficial and long course on the side of the neck and the lateral aspect of the chest. Most commonly paralysis of the serrate muscle is of **traumatic origin,** as, for instance, from punctured or incised wounds of the neck, or from the carrying of heavy weights upon the shoulder. At times paralysis of the serrate muscle results from **muscular over-exertion.** I have seen such a condition after the mowing of hay and after long-continued weaving. Paralysis of the serrate muscle has been observed also after too long-continued planing and sawing. *Infectious paralysis of the serrate muscle* has been reported in the sequence of typhoid fever, diphtheria, erysipelas, and other infectious diseases. *Refrigeratory paralysis of the serrate muscle* is also known. Almost always the

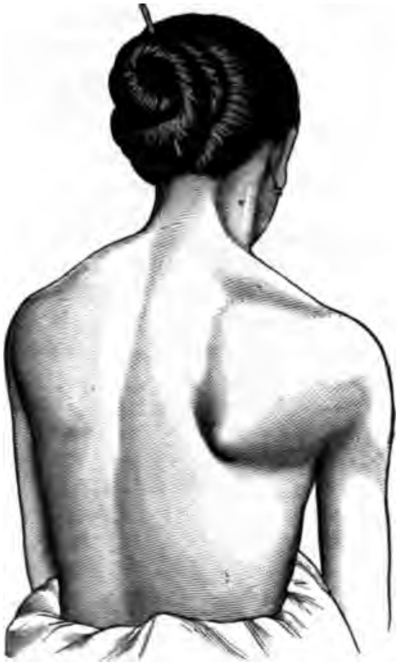


FIG. 68.—Position of the scapula with the arm dependent in a case of paralysis of the right serrate muscle in consequence of muscular over-exertion in a weaver for fourteen years (personal observation, Zurich clinic).

condition occurs in *adults,* and more commonly in *men* than in women.

Symptoms and Diagnosis.—Paralysis of the serrate muscle is attended with an **abnormal position of the scapula,** which is apparent even with the arm at rest and dependent, but becomes more conspicuous when the arm is held in front of the body or is elevated vertically. When the arm is dependent the **inferior angle of the scapula** is removed from the dorsal aspect of the chest (from preponderant action of the biceps, the lesser pectoral, and the coracobrachial). The **inner border of the scapula** is more closely approximated to the vertebral column upon the diseased than upon the healthy side (from preponderant action of the rhomboid and the trapezius), and the inferior angle of the scapula is more closely approximated to the vertebral column than the upper and inner angle. In consequence, the inner border of the scapula is directed

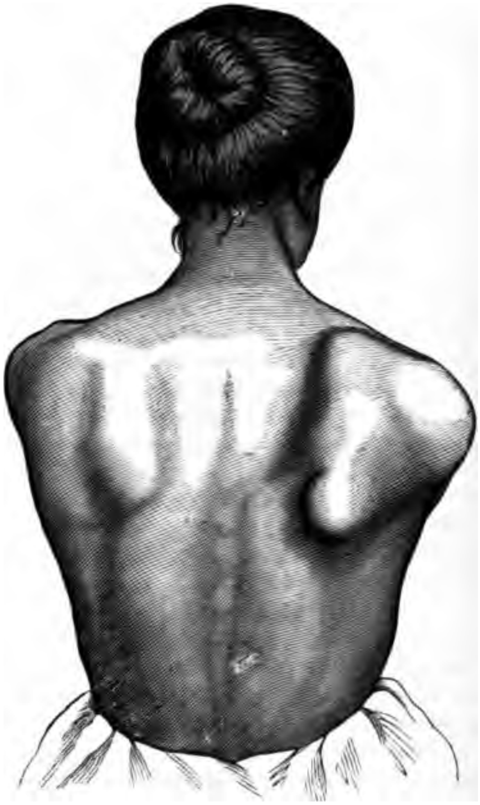


FIG. 69.—The same patient as shown in Fig. 68, with the arm elevated in front.

from above and without downward and inward. The entire scapula occupies a higher level upon the diseased than upon the healthy side (Fig. 68). If the arm is raised in front of the body, the internal border of the scapula becomes so greatly removed from the dorsal aspect of the chest that the hand can be easily inserted between the chest-wall and the border of the scapula (Fig. 69). At the same time the scapula becomes still more closely approximated to the vertebral column. This approximation becomes the greater if the arm is raised to a vertical position (Fig. 70). Elevation of the arm slowly above the horizontal level is possible only with difficulty, and can be effected with greater ease only if the scapula has previously been put in proper position by the hands and is there held during the act of elevating the arm. All movements of the arm forward (pushing, crossing) are rendered difficult, and the patient is unable to offer any resistance to retraction of the scapula. Gradually the serrate muscle may undergo

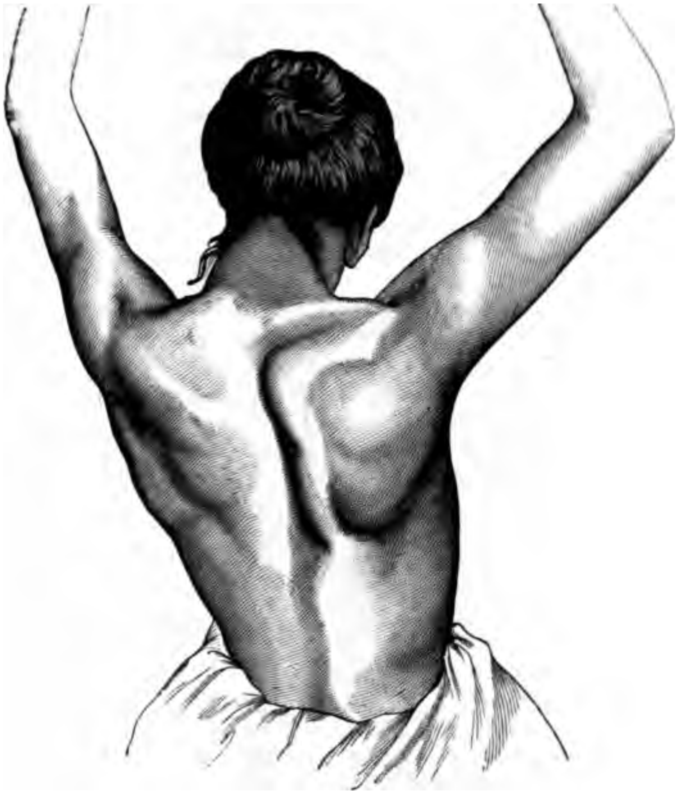


FIG. 70.—The same patient as shown in Figs. 68 and 69, with the arms elevated vertically.

atrophy from disuse, the thorax becoming flattened on its lateral aspect and the otherwise readily visible serrations of the muscle being lost. As a **prodrome** of paralysis of the serrate muscle, pain at times appears in the shoulder, and subsequently may be replaced by cutaneous anesthesia. Paralysis of the serrate muscle is generally unilateral. Traumatic bilateral serrate paralysis may be observed if heavy weights are carried first upon one shoulder and, after paralysis has developed, subsequently upon the opposite shoulder, or if at first one of the muscles and then the other is unduly strained.

PARALYSIS OF THE GREATER AND LESSER PECTORAL MUSCLES.

The greater and lesser pectoral muscles are supplied by the anterior thoracic nerve. Paralysis of these muscles is uncommon, and is characterized by impairment of adduction of the upper arm upon the paralyzed side, and absence of muscular elevation in the infraclavicular region on attempted adduction. The patient is able only with difficulty to place the hand upon the healthy shoulder, and to clap the hands. Little resistance is offered to passive abduction of the upper arm. Atrophy of the paralyzed muscles is attended with the development of a marked depression in the infraclavicular region.

PARALYSIS OF THE RHOMBOID AND THE ELEVATOR OF THE ANGLE OF THE SCAPULA.

The rhomboid and the elevator of the angle of the scapula are supplied by the dorsal nerve of the scapula from the brachial plexus. Derangement of function appears in connection with paralysis of the muscles named only when the trapezius is paralyzed at the same time. Under such conditions paralysis of the rhomboid prevents approximation of the scapula to the vertebral column, while paralysis of the elevator of the scapula prevents movement of the scapula upward.

PARALYSIS OF THE BROAD DORSAL MUSCLE.

Paralysis of the broad dorsal muscle, supplied by the subscapular nerves of the brachial plexus, is attended with difficulty in adduction of the arm to the chest, but particularly in the movement of the arm and the hand toward the gluteal region.

PARALYSIS OF THE INTERNAL ROTATORS OF THE ARM.

The internal rotators of the arm include the *subscapular* and the *greater round* muscle, both of which are supplied by the subscapular nerves of the brachial plexus. When the muscles named are paralyzed the action of the external rotators of the arm (infraspinous, lesser round muscle) preponderates, so that the palmar aspect of the arm and the hand is directed forward, and the arm cannot be rotated inward on request. All of the movements of the hand directed to the opposite side of the body are, therefore, rendered difficult. If atrophy of the subscapular occurs, crackling sounds are audible on movement of the scapula, but these are present also in some healthy persons.

PARALYSIS OF THE EXTERNAL ROTATORS OF THE ARM.

The external rotators of the arm, the *infraspinous* and the *lesser round* muscle, are supplied by different nerves, namely, the infraspinous by the suprascapular and the lesser round by the axillary. Paralysis of both muscles is attended with internal rotation of the arm from preponderant action of the internal rotators, so that the ulnar border of the arm is directed forward. External rotation of the arm is impossible. When the infraspinous muscle upon the right side is paralyzed the movements necessary in writing and sewing are greatly interfered with. Atrophy of the infraspinous muscle gives rise to a depression in the infraspinous fossa.

PERIPHERAL PARALYSIS OF THE MUSCLES OF THE BACK.

Paralysis of the lumbar extensor muscles is attended with marked convex curvature of the vertebral column forward in standing—*lordosis*—a vertical line through the dorsal vertebræ passing far behind the sacral promontory. In the sitting posture, however, marked *kyphosis* develops in the lumbar region, and the upper portion of the body inclines so far forward that the patient is compelled to support his arms upon the thighs in order not to fall over. In the dorsal decubitus the kyphosis disappears. If the patient be seated upon the floor and attempts to arise, he will slowly climb up upon his own extremities with rotatory movements of the body. The waddling, duck-like walk is, further, peculiar. *Paralysis of the extensors of the back* is attended with paralytic kyphosis in the standing posture, which disappears in the dorsal decubitus. Unilateral paralysis will give rise to paralytic scoliosis. *Paralysis of the extensors of the neck* is attended with drooping forward of the head.

PERIPHERAL PARALYSIS OF THE ABDOMINAL MUSCLES.

Unilateral paralysis of the abdominal muscles is attended with retraction of the umbilicus toward the healthy side with each expiration. *Bilateral paralysis of the abdominal muscles* prevents

all expulsive and expiratory movements (cough, sneezing, strai
ing at stool). The hand applied to the abdominal wall fails
detect contraction during the movements named. Change fr
the recumbent to the sitting posture is possible only with the a
of the arms. In standing, the abdomen protrudes greatly f
ward. In walking, the upper portion of the body is bent forwa
and the lumbar portion of the vertebral column is the seat
lordosis. A vertical line through the cervical vertebræ will p
in front of the sacral promontory, as the pelvis is bent grea
forward. *Simultaneous paralysis of the abdominal muscles a*
the muscles of the back is attended with impossibility of maintai
ing the upright posture.

PERIPHERAL PARALYSIS OF THE CRURAL NERV

Paralysis of the crural nerve of refrigeratory, toxic, or inf
tious origin is uncommon; most frequently it is of *trauma*
origin. The causes may reside within the vertebral canal (hem
rhage) or consist in disease of the vertebral column (fractu
tuberculosis, new-growth) or inflammation or a new-growth
the true pelvis. Aneurysms of the femoral artery and punctur
and incised wounds may also be mentioned as causes for the par
ysis. Paralysis of the *iliopsoas muscle* prevents flexion of t
thigh upon the abdomen, and paralysis of the *quadriceps exten*
of the thigh renders impossible extension of the flexed thigh
resistance to passive flexion. Naturally, the *patellar tendon-ref*
is wanting. Such *sensory disturbances* as may be present are d
tributed upon the anterior and inner aspect of the thigh, and
the course of the greater saphenous nerve, also along the inn
aspect of the leg and the inner border of the foot. Atrophy
the paralyzed muscles from disuse is attended with wasting
the thigh upon its anterior aspect. The paralysis of the musc
named is attended further with disturbances in walking and
changing from the sitting to the standing posture.

PERIPHERAL PARALYSIS OF THE OBTURATOR NERVE.

Peripheral paralysis of the obturator may result from the sa
causes as paralysis of the crural nerve, and at times both ner
are paralyzed simultaneously. Occasionally paralysis of the
turator nerve is observed after difficult labor or after incarce
tion of an obturator hernia. The abductors of the thigh parti
larly are paralyzed, so that the patient is unable to move
abducted member to the middle line, or to throw it over
healthy member in the recumbent or sitting posture. In con
quence of paralysis of the external obturator, external rotation

the lower extremity also is embarrassed. Sensory disturbances may be present upon the internal aspect of the thigh.

PERIPHERAL PARALYSIS OF THE GLUTEAL NERVES.

Paralysis of the gluteal nerves is attended with difficulty in *rotation of the thigh* both inward (middle and lesser gluteus, internal obturator, tensor of the broad fascia) and outward (greater gluteus). *Abduction of the leg* is interfered with (greater and middle gluteus). *Walking*, particularly ascending stairs, is rendered difficult, because the iliopsoas and the greater gluteus are compelled to fix the trunk upon the thigh and to maintain the equilibrium, and the action of the iliopsoas preponderates in the presence of paralysis of the greater gluteus. In consequence of paralysis of the greater gluteus change from flexion of the body forward to the upright posture is impossible. Atrophy of the gluteal muscles from disuse is characterized by flattening and wasting of the buttocks.

PERIPHERAL PARALYSIS OF THE SCIATIC NERVE.

Etiology.—Peripheral paralysis of the sciatic nerve may involve the trunk of the nerve or individual branches. In the latter event peroneal is more common than tibial paralysis. The *trunk of the sciatic nerve* often suffers from *traumatism*, while refrigeratory, infectious, and toxic paralyses are observed much less commonly. The traumatic causes are approximately the same as those for peripheral paralysis of the crural nerve. *Isolated palsies of the peroneal nerve* have been observed in a number of instances in laborers compelled to occupy a stooping posture for a long time in their work (potato-pickers, stone-setters, asphalters).

Symptoms and Diagnosis.—In cases of paralysis of the trunk of the sciatic nerve **flexion of the leg upon the thigh is impossible** (paralysis of the biceps of the thigh, the semitendinosus, the semimembranosus). External rotation (internal obturator, gemelli, pyriform) and abduction of the thigh also are restricted. In walking the extremity remains straight and is not flexed at the knee-joint. In the presence of *peroneal paralysis* the tip and the outer border of the foot are directed downward (pes varo-equinus). In walking the patient must lift the foot high, in order to avoid scraping the floor with the toes, and the foot is brought down upon its outer border. Dorsal flexion of the foot and the toes is impossible. *Paralysis of the tibial nerve* prevents plantar flexion of the foot and the toes, and, from the preponderant action of the antagonists, gradually gives rise to *pes calcaneus*—club-foot. In addition, in consequence of paralysis of the posterior tibial muscle, abduction of the foot is greatly impaired (posterior tibial).

PERIODIC PARALYSIS.

The designation periodic paralysis has been applied to a condition in which persons are periodically paralyzed for hours in th upper and lower extremities, and at times also in a number o cerebral nerves. The electric irritability of the paralyzed nerve and muscles is greatly diminished, or even at times abolishe The disease is hereditary. It has been thought to be dependen upon auto-intoxication.

SPASMODIC DISORDERS OF MOTOR NERVE (HYPERKINESIS).

Muscular spasm may be either **tonic** or **clonic.** With toni spasm there is persistent contraction of the muscle, which may remain shortened and hard for hours and at times for days. Cloni spasm is attended with intermittent muscular contractions usuall appreciable to the eye and the applied hand and capable of enumeration. If tonic muscular spasms are attended with pain, the are designated *cramps.*

MOTOR TRIGEMINAL SPASM.

Motor trigeminal spasm is known also as *masticatory spasm,* because it involves the muscles of mastication. *Tonic spasm of th muscles of mastication* gives rise to the clinical picture of *trismus* o *lockjaw.* The jaws are clinched tightly and the patient is unabl to separate them, speaking through the teeth, and the contracte temporals and masseters are usually visible and palpable beneat the skin as muscular masses of board-like hardness. Naturall persistent trismus is attended with the danger of starvation, s that it becomes necessary for purposes of artificial nutrition to introduce an esophageal tube between the teeth or into the nose, or t administer nutrient enemata. Most commonly trismus results i consequence of reflex irritation from the irruption of teeth an periostitis of the lower jaw. It is quite a constant symptom o tetanus, although here it is probably due to central irritatio The best-known examples of *clonic spasm of the muscles of mastication* are the *chattering of the teeth* on exposure to cold and t emotional influences, and the *grinding of the teeth* in cases of meningitis and in teething children. In the first event particularl the masseters and the temporals are involved, in the latter th pterygoids. At times spasm of the muscles of mastication is associated with trigeminal neuralgia.

In the **treatment** an attempt will be made first to remove th causative factors. In addition, subcutaneous injections of morphi applications of the galvanic and the faradic current, and in the presence of trismus even gradual separation of the jaws by the introduction of wedges.

SPASM OF THE MUSCLES OF THE FACE (PSOROSPASM).

Symptoms, Diagnosis, and Prognosis.—Most commonly **clonic spasm of the muscles of the face** is observed. If *all of these muscles* are involved, the condition is generally **unilateral.** Spontaneously and particularly on emotional disturbance active contractions take place in the various muscles of the face, giving rise to marked and disfiguring grimaces. Wrinkling of the forehead, blinking of the eyes, distortion of the cheeks, puckering of the lips, and dilatation of the mouth alternate irregularly with one another, and the appearance resembles the distortions of the facial muscles induced by faradization of the facial nerve. At times **pressure-points** are discovered over the supraorbital or the infraorbital nerve, the spinous or the transverse processes of the cervical vertebræ, the condyles of the radius or the ulna, the mucous membrane of the buccal or the nasal cavity, pressure upon which causes cessation of the spasm. The number of paroxysms of spasm varies; often they are repeated within a few minutes. The individual paroxysm is of widely varying intensity, extent, and duration. At times there is but a transitory, lightning-like contraction of the muscles of the face. The stapedius, the muscles of the palate, and the salivary secretory nerves do not, as a rule, participate in the clonic muscular spasm. The contractions usually cease during sleep, although I have observed them to persist in cases of cerebral hemiplegia. The disorder is annoying, but not dangerous. Not rarely it persists throughout life. Recovery can be expected only when the causative factors are discoverable and are capable of removal.

Partial clonic spasm of the muscles of the face occurs also in healthy persons following great mental excitement, as, for instance, involuntary wrinkling of the forehead, the play of the alæ of the nose, and the twitching of the lips. The best-known of the varieties of partial clonic spasm of the facial muscles is clonic spasm of the sphincter of the eyelids—*nictitation*—which is observed with particular frequency in association with diseases of the eyes themselves.

Tonic spasm of the facial muscles is most commonly partial, and involves with preference the sphincters of the eyelids. With the onset of such spasm—*blepharospasm*—the eyelids snap together like the lid of a chest, and remain closed until the attack terminates. The duration of an attack varies between minutes, hours, and weeks. Naturally, the patient is unable to see during the attack, and if this occur on the crowded street the patient may be exposed to serious danger. It is noteworthy that at times points are discoverable upon the body, as, for instance, over the supraorbital and infraorbital nerves, the cervical vertebræ or elsewhere, on pressure upon which the muscular spasm ceases and the closed eyelids separate. The disorder may be extremely obstinate and persist throughout the whole of life.

Etiology.—Spasm of the facial muscles is observed with particular frequency in **nervous persons.** At times the disorder is **inherited,** although some members of the family perhaps will not suffer from spasm of the facial muscles, but from certain central neuroses (hysteria, neurasthenia, epilepsy, chorea, psychopathy, etc.). At times the disorder is due to traumatism. Thus, it has been observed to develop in the sequence of contusion of the trunk of the facial nerve, as a result of the pressure exerted by enlarged lymphatic glands or by an aneurysm of a cerebral artery. Frequently it occurs in consequence of **reflex irritation,** as, for instance, in association with trigeminal neuralgia, and diseases of the eyes and the teeth. It has been observed also in women with disease of the uterus or the ovaries, and in persons with intestinal worms. In children the **habit of imitation** plays an important part; that is, previously healthy persons who are brought much in contact with those presenting facial spasm are attacked by the same disorder. Also originally voluntary **grimaces** may gradually be converted into involuntary facial spasm. Experience has shown that *men* are attacked by spasm of the facial muscles more commonly than women. The disorder occurs less commonly in childhood.

Anatomic Alterations.—But little is known with regard to the anatomic alterations attending spasm of the facial muscles. In any event the latter may occur in the absence of any change in the fibers of the facial nerve, so that the disturbances present must be of a purely functional nature.

Treatment.—In the first place, an effort should be made to remove any causative factors present—**causal therapy**—consideration being given particularly to facial spasm of reflex origin. The rapidity with which the spasms cease after removal of the causative conditions is often surprising. Unfortunately, such cases are the less common. As a rule, one is compelled to resort to **symptomatic treatment.** In nervous persons success may be attained by means of a *judicious mode of life* and the administration of *nervines,* particularly *bromids. Narcotics* also, particularly subcutaneous injections of morphin, have been largely used. At times **psychic treatment** will be indicated, particularly in children. If *pressure-points* can be demonstrated, applications of the galvanic current through the anode should be made to them. Recourse has been had also to **nerve-section (neurotomy), nerve-excision (neurectomy),** and **nerve-**

stretching; but generally the twitchings cease only so long as the facial paralysis persists, generally recurring with the disappearance of the paralysis.

SPINAL ACCESSORY SPASM.

What has been said concerning spasm of the facial muscles is applicable also to the *causes* of spinal accessory spasm. At times *disease of the cervical vertebræ* is responsible for the disorder. In children it has been observed at the period of *dentition*. *Unilateral clonic spasm of one sternomastoid* is attended with periodic twitching movements of the head, the face and the chin being rotated toward the healthy side, the chin elevated, and the occiput retracted. *Unilateral tonic spasm of the sternomastoid* is attended with persistence of this position during the attack and immobility of the head—spasmodic torticollis. *Bilateral clonic spasm of the sternomastoid* is attended with various manifestations, accordingly as the spasmodic muscular contractions of the sternomastoid occur alternately or synchronously. In the first instance the head is involved in rotatory movements, in the latter in nodding movement. *Bilateral tonic spasm of the sternomastoid* is attended with retraction of the head.

In addition to the sternomastoid, the *trapezius* also is involved in cases of accessory spasm. Unilateral clonic spasm of the trapezius is attended with elevation of the scapula and retraction of the head. The symptoms of bilateral clonic as well as of tonic spasm of the trapezius may be understood without further description. A striking and varying picture of alterations in the movements of the head and the shoulder results when both the sternomastoid and the trapezius are involved in the spasm, although it is easy to separate the picture into its individual constituents. With regard to the prognosis and the treatment, the same statements are applicable as were made concerning spasm of the facial muscles.

HYPOGLOSSAL SPASM.

Hypoglossal spasm is attended with *spasm of the tongue—glossospasm*—and accordingly as this occurs in the act of chewing or of speaking a distinction is made between *masticatory* and *articulatory spasm of the tongue*. The spasm may be clonic or tonic and unilateral or bilateral. The disorder can be readily recognized, and attacks particularly hysterical and nervous persons. In accordance with the character of the spasm, either the masticatory or the articulatory movements are interfered with. If the tongue is retracted for a considerable length of time, danger of

SPASM OF THE CERVICAL AND SCAPULAR MUSCLES.

Spasm of the Inferior Oblique Muscle of the Head.—This muscle rotates the head in the horizontal level, without elevating or depressing the chin. If the disorder be unilateral and clonic, the head is rotated horizontally toward the diseased side, while in the presence of bilateral alternating muscular spasm rotatory movements of the head from side to side occur—*rotatory spasm* or *tic.* Unilateral tonic spasm keeps the head constantly turned toward one side, and resistance is encountered when an attempt is made to restore the head to its normal position.

Spasm of the splenius muscle of the head also gives rise to an abnormal position of the head. The muscle turns the head toward the diseased side, while the face and the chin are approximated to the shoulder. The contracted muscle is visible beneath the anterior border of the trapezius, and the sternomastoid appears relaxed upon the diseased side and stretched upon the healthy side (Fig. 71).

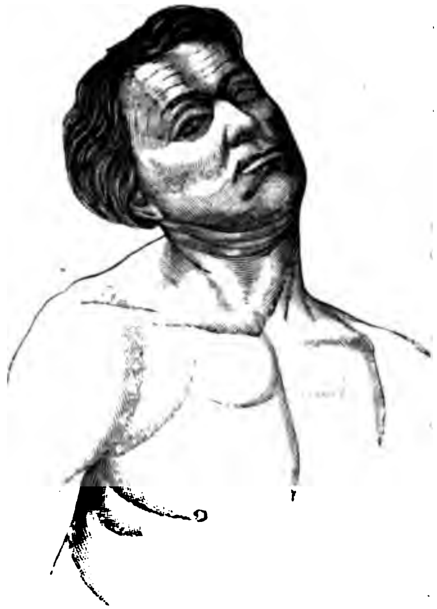


FIG. 71.—Position of the head in the presence of spasm of the right splenius muscle of the head.

When **spasm of the rhomboid** is present the scapula becomes approximated to the vertebral column, and cannot be moved freely outward. Elevation of the arm above the horizontal level is attended with resistance. The contracted muscle is visible and palpable in the interscapular space.

Spasm of the elevator of the scapula is attended with elevation of the scapula, particularly its inner upper angle. The head is retracted. The contracted muscle appears in the clavicular fossa at the side of the anterior border of the trapezius.

SPASM OF THE DIAPHRAGM.

Clonic spasm of the diaphragm, also designated *hiccough*, is the best-known variety. While the diaphragm contracts suddenly and air endeavors to enter the air-passages, the chink of the glottis closes and then a gurgling sound is heard. The condition may come to an end with a few contractions of the diaphragm, but in some cases it persists for days and weeks, and the patient complains of pain and disagreeable sensations along the points of attachment of the diaphragm. *Children* are predisposed in marked degree to clonic spasm of the diaphragm, which may occur after overloading the stomach and after hearty laughter. The same causes may give rise also to clonic spasm of the diaphragm in adults. At times the disorder occurs in association with *diaphragmatic pleurisy* or *peritonitis*, or with *irritation of the phrenic nerve by mediastinal tumors. Disease of the brain* (meningitis, hemorrhage, softening) also is at times attended with spasm of the diaphragm. Occasionally this occurs in cases of *hysteria. Reflex clonic spasm of the diaphragm* has been observed in connection with diseases of the stomach, the intestine, the kidneys, the liver, and the sexual organs. Further, there are cases *without obvious etiologic factors*.

In **treatment** efforts should be directed first to the removal of the cause—**causal therapy.** Among **symptomatic measures** expulsive efforts with closure of the chink of the glottis, rapidly successive swallowing of water, the introduction of a sound into the esophagus, constriction of the lower portion of the thorax with simultaneous strong flexion of the head, and the like, have been recommended. Among medicaments, **nervines** and **narcotics** particularly have been employed. In cases of hysteria **psychic treatment** is indicated. I have effected cure by means of regular and slow counting.

Tonic spasm of the diaphragm has been observed as a result of exposure to cold and in the sequence of infectious diseases (particularly rheumatism, tetanus). At times it is dependent upon disease of the brain (epilepsy, hysteria). It may be recognized from the want of movement in the greatly distended lower portion of the thorax in respiration, while the upper portion is engaged in active and labored breathing. The lower border of the heart and of the lungs is unusally low, and exhibits no movement with respiration. Respiratory movement of the lower border of the liver is likewise not demonstrable. The epigastrium bulges forward. If speedy relief be not afforded, death will result from suffocation. A subcutaneous injection of morphin should be given or chloroform-narcosis be induced in order to overcome the tonic spasm of the diaphragm. If these measures do not succeed, *faradization of the phrenic nerves* should be practised.

SPASM OF THE ABDOMINAL MUSCLES.

Spasm of the abdominal muscles is generally of central origin. Clonic spasm of the abdominal muscles has occasionally been observed in connection with hysteria; tonic spasm—recognizable from the hardness, tension, and contraction of the abdominal walls—occurs in connection with tetanus and meningitis.

MUSCULAR SPASM IN THE UPPER AND THE LOWER EXTREMITIES.

The muscles of the arms and the legs are but rarely involved in tonic or clonic spasm. The condition when present can be readily recognized.

CRAMPS.

The best-known example of tonic, *painful* muscular spasm is cramp in the calf of the leg. The patient complains suddenly of intense pain in the calf of one leg and at the same time the muscles in this situation are thickened and contracted with board-like hardness. As a rule, the pain ceases in the course of a few seconds, although a sense of weakness persists for some time. Occasionally extravasations of blood beneath the skin are observed in the region of the calf. *Nervous* and *anemic individuals* are greatly predisposed to the disorder. At times it develops in the sequence of *over-use* on the part of the calf-muscles in standing, walking, swimming, turning, dancing. The cramps in the calves that are observed in cases of Asiatic cholera and diabetes mellitus appear to be dependent upon *toxic influences*. Cramps in the calves develop not rarely in persons with *varicose dilatation of the veins of the leg*. The cramp can be overcome by vigorous *rubbing*, by *alcoholic frictions*, and by subcutaneous *injection of morphin*. Besides, attention should be given to the *causative conditions*.

NEURALGIA.

PRELIMINARY CONSIDERATIONS.

Neuralgia—nerve-pain—is characterized by its distribution in the course of diseased sensory nerves. Either the pain occurs in attacks or it is greatly increased paroxysmally. The number and the duration of the attacks of pain are susceptible of wide variation. The pain is described by the patient as gnawing, burning, and crushing, and not rarely becomes so intense that even strong men are made to groan and are incapacitated from all physical and mental occupation. Not rarely rest at night is greatly disturbed. The *pressure-points* of Valleix are peculiar to many varieties of neuralgia. It has been found that certain points in the course of the diseased nerve are particularly sensitive to pressure. The pressure-points are definite for the individual nerves, being generally situated at places where the nerve is superficial and rests upon bone, so that it is readily accessible to the compressing finger. *Cutaneous sensibility* in the distribution of the diseased nerve is frequently altered. At the beginning there is frequently hyperesthesia, which is subsequently followed by anesthesia. At times *vasomotor*, *secretory*, and *trophic disturbances* are present. It may also happen that severe pain radiates throughout an extensive cutaneous area, and it may give rise to *clonic* or *tonic muscular spasm*.

Anatomic alterations may be wanting in the nerve even though the neuralgia have lasted for years, while in other instances neuritic changes are found; and accordingly a distinction can be made between *neurotic* and *neuritic neuralgia*. Further, the differential diagnosis generally is extremely difficult during life, and often impossible.

Among the *causes for neuralgia* are the same conditions that have been named as causes for peripheral paralysis, and accordingly *refrigeratory* (*rheumatic*), *traumatic*, *infectious*, and *toxic neuralgia* may be distinguished. It has also been maintained that *reflex neuralgia* occurs, developing in the sequence of antecedent disease in remote organs, particularly in the female generative organs. Such nerves as lie superficially and pursue a long course or pass through narrow bony canals are readily exposed to injury. The sciatic and the trigeminus are, therefore, attacked by neuralgia with particular frequency. Among the infectious forms of neuralgia the *malarial* variety is especially well known, and this may be the sole manifestation of malarial infection, and is characterized by its intermittent occurrence. It is remarkable that typically recurring neuralgia, so-called masked intermittent, may occur in regions free from malaria and attack persons who have never lived in malarial regions, so that doubt as to the malarial nature of some cases of intermittent neuralgia appears justified, doubt that may perhaps be removed by examination of the blood for malarial plasmodia. In addition to malaria, *syphilis* is yet a common cause for neuralgia. Among the toxic varieties of neuralgia may be included the *uremic*, the *gouty*, and the *diabetic*, and which more appropriately may be designated autotoxic. Experience has shown that members of nervous families and pale, nervous, debilitated individuals are attacked by neuralgia with particular frequency and readiness. Women are more greatly predisposed to neuralgia than men.

The *curability of neuralgia* depends essentially upon its causes. At times it persists throughout life, and embitters the existence of the patient to such a degree as to induce suicide.

The **treatment** *of neuralgia* should be, in the first place, causal. Should exposure to cold have been operative, resort may be had to diaphoretics, hot fomentations, applications of cotton batting, and the internal administration of salicylic acid or sodium

salicylate (1.0—15 grains—every two hours), of antipyrin (1.0—15 grains—thrice daily), or phenacetin (1.0—15 grains—thrice daily). When syphilis is the etiologic factor inunctions of mercurial ointment (5.0—75 grains—daily), and internally potassium iodid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—one tablespoonful—thrice daily) may be prescribed. Malarial influences will indicate the employment of quinin hydrochlorate (from 1.0 to 5.0—from 15 to 75 grains—to be given three hours in advance of the expected paroxysm). At times surgical intervention will be required for the removal of the cause of neuralgia, as, for instance, in the removal of a tumor exerting pressure upon a nerve, and the like. Among **symptomatic measures** subcutaneous *injection of morphin* is the most reliable, but in cases of obstinate neuralgia there will be risk that the patient will demand progressively increasing doses, and become a morphin-habitué, particularly if he be entrusted with a syringe and a solution of morphin:

℞ Morphin hydrochlorate, 0.3 (4½ grains);
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: From 0.25 to 0.5 (4 to 8 minims) subcutaneously.

Even when malarial infection is not suspected quinin hydrochlorate (from 1.0 to 5.0—from 15 grains to 75 grains—in tablet) is a useful remedy. In addition, **nervines** and **narcotics** of all kinds have been employed. It will suffice to mention a few examples from which success may most likely be expected:

℞ Bitter-almond water,
Solution of potassium arsenite, each, 5.0 (75 minims).—M.
Dose: 10 drops thrice daily after eating.

℞ Atropin sulphate, 0.01 (⅙ grain);
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: 0.25 (4 minims) subcutaneously.

℞ Solution of cocain hydrochlorate, 0.5 : 10.0 (7½ grains : 2½ fluidrams).
Dose: 0.5 (8 minims) subcutaneously.

℞ Exalgin, 0.3 (4½ grains);
Sugar, 0.5 (7½ ").—M.
Make 10 such powders.
Dose: 1 powder thrice daily.

℞ Tincture of gelsemium, 20.0 (5 fluidrams).
Dose: 10 drops thrice daily.

℞ Butyl chloral hydrate, 5.0 (75 grains);
Glycerin, 20.0 (5 fluidrams);
Distilled water, 130.0 (4 fluidounces).—M.
Dose: 15 c.c. (1 tablespoonful) every five or ten minutes until half the prescription is used.

Alcoholic frictions and **narcotic** and **irritating ointments** have **also been considerably** used. It is a matter of indifference with regard to the results whether spirit of mustard, spirit of camphor, spirit of formic acid, or compound spirit of angelica be employed. Chloroform-liniment also may be used:

℞ Chloroform,	10.0 (2½ fluidrams);
Ammoniated liniment,	40.0 (10 ").—M.
Apply with friction.	

The following examples of *narcotic* and *irritating ointments* may be given:

℞ Morphin hydrochlorate,	0.1 (1½ grains);
Wool-fat,	
Lard,	each, 5.0 (75 ").—M.
For inunction.	

℞ Extract of belladonna,	2.0 (30 grains);
Wool-fat,	
Lard	each, 5.0 (75 ").—M.
For external application.	

℞ Veratrin,	0.3 (4½ grains);
Wool-fat,	
Lard,	each, 25.0 (6 drams).—M.
For inunction.	

Leeches, wet cups, and applications of ice to the nerve and hot fomentations have also been employed considerably. Some patients are greatly relieved by the employment of the **galvanic current.** The current should not be too strong and the anode (+ pole, copper-pole) should be applied to the affected nerve. The application should be repeated daily, but should not be continued longer than from three to five minutes. **Treatment with the faradic brush** may also yield good results. In obstinate and distressing cases resort has been had to **surgical measures,** and nerve-stretching, nerve-section (neurotomy), nerve-resection (neurectomy), and recently also the extraction of exposed nerves by means of forceps have been practised. Unfortunately, all of these efforts are attended either with no result or too frequently with only a transient result. Any effect will be wanting if the operation fails to reach the seat of the neuralgia, and this may happen all the more readily as no remedy is known that is capable of determining with certainty the seat of neuralgia. Neuralgia cured by operation recurs readily after the divided or exsected nerve has united or reformed, and this may happen even when remarkably long portions of the nerve have been removed. Great care should therefore be exercised in promising a successful result.

TRIGEMINAL NEURALGIA.

Etiology.—Trigeminal neuralgia is one of the *commonest varieties of neuralgia,* as the branches of the trigeminal nerve pass through several bony canals and in part lie superficially, so that they may be readily affected by injurious influences. *Refrigeratory trigeminal neuralgia* occurs with certainty, and it may develop, among other conditions, when individual branches are exposed to a draft of cold air or to rain. Among the forms of *infectious neuralgia* the best known is that of malarial origin. Trigeminal neuralgia, however, occurs not at all rarely in connection with typhoid fever, syphilis, and influenza. *Toxic trigeminal neuralgia* has been observed as a result of the action of lead, mercury, and iodin, and in cases of gout and of diabetes. *Traumatic trigeminal neuralgia* occurs frequently. Among the causes, contusions and stab-wounds of the nerve, periostitis of the cranial bones, compression by a neoplasm, the extension of neoplastic proliferation into the trunk of the nerve, inflammatory processes in the teeth or the jaw, in the nose or in the frontal sinuses, and in the eye and the ear. At times trigeminal neuralgia is dependent upon disease at the base of the skull (syphilitic inflammation and thickening of the meninges, tumors, aneurysms of the cerebral arteries). Children generally are exempt from trigeminal neuralgia, excepting alveolar neuralgia (toothache). Women are attacked more frequently than men, and pale and nervous persons in greater number than the robust.

Symptoms and Diagnosis.—Trigeminal neuralgia is, as a rule, **unilateral.** Naturally, after the lapse of a certain time it may gradually extend to the opposite trigeminal nerve, while the nerve first involved becomes free from pain. Only rarely are all three branches of the trigeminal nerve simultaneously attacked by neuralgia, and such a circumstance must be indicative of a basal origin, as the branches lie close together in this situation. Most commonly only a few branches of the trigeminal nerve are affected by neuralgia. Of all of the branches of the trigeminal nerve the supraorbital is most commonly attacked.

At times the neuralgia is preceded by certain paresthesias (a sense of crawling or of coldness or of stiffness) as **prodromes.** The **pain** may possess a burning, boring, or crushing character, and at times attains an unendurable intensity. The duration of an attack of pain and the frequency of its recurrence vary extremely. The individual attack of pain sets in at times without recognizable cause, but at other times it is excited by emotional disturbances, a draft of air, glaring light, eating, drinking, or the like. In the course of the affected nerve Valleix's **pressure-points** can generally be demonstrated. **Cutaneous sensibility** is at first generally heightened, but subsequently becomes diminished or, less commonly, lost. **Vasomotor disturbances** are at first indicated by arterial spasm and pallor of the skin, and soon vascular dilatation occurs, with corresponding redness and warmth of the skin. Among **secretory disturbances** increased secretion of tears, of nasal mucus (at times bloody), of saliva, and of perspiration has been observed. **Trophic disturbances** are less common, and they may consist in increase or diminution in the fatty layer of the skin, coarseness, graying, and loss of the hair, eruptions of herpes, iritis, glaucoma, or neuroparalytic ophthalmia. Severe attacks of pain are at times attended with clonic muscular spasm in the face and at times also in the extremities. When the causative conditions are incurable the disorder may persist for many years, and even throughout the whole of life. The fear of an attack of neuralgia in eating, sleeplessness at night, and seclusion result in physical and moral depression, and may lead to suicide. For those familiar with the anatomic distribution of the trigeminal nerve there is no

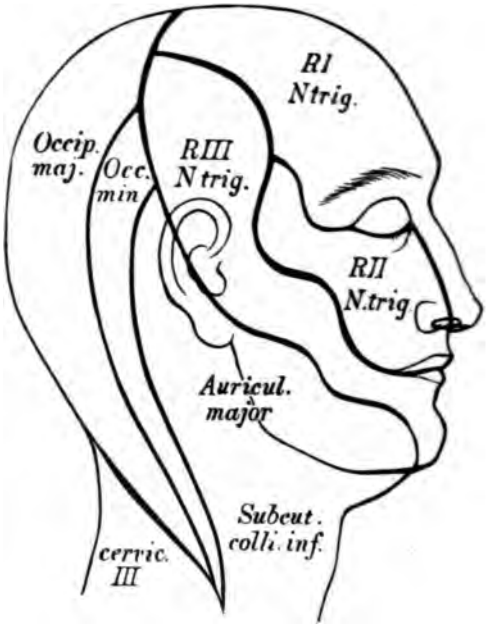


FIG. 72.—Distribution of the trigeminal nerve upon the face.

difficulty in determining with certainty the branch of the nerve that is the seat of neuralgia. For ready recognition reference may be made to Fig. 72, and we shall confine ourselves to the following diagnostic points:

In cases of *supraorbital neuralgia* the pain radiates from the supraorbital margin to the frontal region, toward the root of the nose, and into the upper eyelid. The most constant pressure-point is situated just below the supraorbital foramen in the roof of the orbit.

Infraorbital neuralgia is attended with pain radiating from the infraorbital margin along the cheek to the upper lip and the lateral aspect of the nose. The distinctive pressure-point is at the infraorbital foramen.

Lingual neuralgia or *glossalgia* is attended with severe pain in one-half of the tongue, and often with coating of part of the tongue, together with thickening of this half of the tongue and increased secretion of saliva.

The diagnosis should never be considered complete with the localization of the neuralgia, but in every instance an effort should be made to determine the causative factors.

Prognosis.—The prognosis depends upon the curability of the causative factors.

Treatment.—The treatment is the same as that described on pp. 514–516. In obstinate cases resort has been had to compression and even to ligation of the common carotid artery, but without certainty as to the result.

CERVICO-OCCIPITAL NEURALGIA.

Etiology.—From the cervical plexus, formed from the highest four cervical nerves, five sensory nerves are given off: the greater and the lesser occipital, the great auricular, the inferior subcutaneous of the neck, and the supraclavicular, of which any one may be attacked alone by neuralgia, or in rare instances all may suffer together. Most commonly neuralgia of the greater occipital is present, and the condition is for brevity's sake designated occipital neuralgia. The causes for the varieties of neuralgia under consideration have already been mentioned several times. Occipital neuralgia is observed particularly after injuries to the scalp, and further in association with syphilis and uremia. At times cervico-occipital neuralgia is dependent upon disease of the cervical vertebræ (tuberculosis, tumors) or upon disease of the spinal membranes or of the cervical cord itself.

Symptoms and Diagnosis.—*Occipital neuralgia* is attended with pain radiating from the occiput to the parietal bone. A pressure-point is situated at the point of exit of the occipital nerve between the mastoid process and the atlas. Occasionally there appear at the time of the attack of pain redness of the face, secretion of tears, contraction of the pupils, and vomiting. The hair may fall out and nodules may form beneath the scalp. The duration of the disorder depends upon the nature of the causative factors.

Neuralgia of the lesser occipital nerve is attended with pain in the lateral aspect of the occiput and extending to the ear. A pressure-point is situated at the point of exit of the nerve just behind the mastoid process. In cases of *neuralgia of the great auricular nerve* the patients complain of pain above the mastoid process, upon the posterior aspect of the auricle, and in the parotid gland, while neuralgia of the *inferior subcutaneous nerve of the* [illegible] attended with pain in the anterior, inferior, and median [illegible] of the neck, and *neuralgia of the supraclavicular nerve* [illegible] in the region of the acromion, the shoulder, and the [illegible] of the thorax.

PHRENIC NEURALGIA.

The designation phrenic neuralgia has been given to pain occurring [illegible] usually in the course of the phrenic nerve and at the [illegible] attachment of the diaphragm, and often giving rise to [illegible] in breathing. The occurrence of such a variety [illegible] has not without reason been doubted. Among the [illegible] that have been named are disease of the heart, [illegible] the aorta, disorders of the liver, the kidneys, the in[illegible] the spleen, pleurisy, pericarditis, and peritonitis.

CERVICOBRACHIAL NEURALGIA.

Neuralgia involving the sensory nerves of the brachial plexus [illegible] the eighth cervical and the first dorsal nerve) [illegible] frequently. Generally it is of traumatic origin (contusion, [illegible], incised, punctured, and gunshot wounds, and the [illegible] several nerve-tracts are involved at the same time. [illegible] and also trophic disturbances occur (herpes, [illegible] of hair, glossy fingers). At times muscular [illegible] are superadded to neuralgia.

[illegible] INTERCOSTAL NEURALGIA.

[illegible]. The twelve dorsal nerves, after leaving the vertebral [illegible] intervertebral foramina, divide into poste[illegible] branches. The posterior nerve-roots or *dorsal* [illegible] upon the skin of the back down to the [illegible] seat of neuralgia. The anterior nerve-[illegible] however, are attacked all the more fre[illegible] causative factors, as with other neuralgias, are [illegible] cold, injury, infection, and intoxication. [illegible] should be noted that some cases are depen[illegible] the vertebral column (tuberculosis, carci[illegible] neuralgia may at times be secondary [illegible] intercostal nerves become compressed [illegible] connective-tissue cicatrices. Often intercostal [illegible] followed by herpes zoster. Malaria has [illegible] a causative factor.

[illegible] Diagnosis.—Intercostal neuralgia is gen[illegible] commonly left-sided, and, as a rule, attacks [illegible] nerves at the same time. The nerves between the fifth and the ninth are involved with especial frequency. The pain is characterized by its radiation about one-half of the chest. **Pressure-points** are present close to the vertebral column, in the axillary region, and close to the margin of the sternum, and are designated as vertebral, lateral, and sternal points respectively. **Hyperesthesia** and subsequently **anesthesia** of the skin are observed frequently. Among **trophic disturbances** herpes occurs not rarely. The patient often complains of disturbed sleep and of difficulty in breathing, laughing, sneezing, and coughing, because the pain is excited or intensified by these acts. Accurate limitation of the pain in the course of the intercostal spaces and the absence of pleuritic friction-sounds and of alterations in the ribs readily distinguish intercostal neuralgia from muscular rheumatism, dry pleurisy, and inflammation of the ribs.

Neuralgia of the mammary gland or *mastodynia* also is a variety of intercostal neuralgia, because in addition to the supraclavicular nerves the mammary gland receives branches also from the second to the sixth intercostal nerve. The disorder is most common in women, particularly hysterical and anemic women, and it develops at times in the sequence of injury to the gland or of unduly protracted lactation. At times small indurations—so-called neuralgic nodes—are present in the painful gland. The disorder is attended with attacks of unbearable pain in one breast or in both. At times a colostrum-like fluid is secreted during an attack of pain. The attack occasionally persists for more than an hour. The obstinacy and the severity of the pain have induced some patients to have the gland amputated.

LUMBO-ABDOMINAL NEURALGIA.

Lumbo-abdominal neuralgia includes involvement of the ilio-hypogastric, ilio-inguinal, lumbo-inguinal, and external spermatic nerves, which originate from the four upper lumbar nerves. The painful area extends over the lumbar and gluteal regions, the scrotum or the labia majora, and the anterior aspect of the thigh. A pressure-point is frequently found at the middle of the crest of the ilium. At the time of the attack of pain there may be priapism, discharge of seminal fluid, or leukorrhea. In addition to exposure to cold, disease of the vertebral column, exudates and tumors in the pelvis, and displacements of the uterus particularly give rise to lumbo-abdominal neuralgia.

CRURAL NEURALGIA.

Crural neuralgia is uncommon, and when it occurs is generally of traumatic origin. Disease of the vertebral column, exudates and tumors in the pelvis, aneurysms of the femoral artery, and crural hernias have been named as causes of crural neuralgia. The pains are distributed especially upon the anterior and internal aspect of the thigh, and, in the course of the greater saphenous nerve, along the inner aspect of the leg and the inner border of the foot to the great toe. Pressure-points may be present as follows: A crural point, just below Poupart's ligament, corresponding to the point of exit of the crural nerve; a knee-point on the inner aspect of the knee-joint; a plantar point, in front of the internal malleolus; and a toe-point, at the base of the great toe.

OBTURATOR NEURALGIA.

Obturator neuralgia may set in suddenly in connection with strangulated obturator hernia, and is therefore of diagnostic importance. The pain extends along the inner aspect of the thigh to the knee. Generally the adductors of the thigh are paralyzed at the same time.

NEURALGIA OF THE EXTERNAL CUTANEOUS NERVES OF THE THIGH.

The pain extends upon the outer aspect of the thigh to the knee. I have observed this variety of neuralgia frequently in individuals who have supported one knee upon the other for long periods of time.

SCIATIC NEURALGIA.

Etiology.—Among all of the varieties of neuralgia sciatic neuralgia, also designated *sciatica*, occurs most commonly. The nerve may readily suffer from exposure to cold and traumatism on account of its superficial position and its long course, but infectious and toxic neuralgia of the sciatic nerve is also known. Working in the damp (as in digging canals, working in the woods or in fortresses, sleeping in damp rooms and in the open air, and the like) is often followed by sciatica. Among the **traumatic causative factors** may be mentioned a fall, a blow, or contusion involving the gluteal region, heavy lifting and long standing, fractures and luxations of the thigh, neoplasms and exudates in the vicinity of the sciatic nerve, displacements and neoplasms of the uterus and the ovaries, parametric and perimetric exudates, pregnancy, fecal accumulation in the rectum, tumors of the pelvic bones, luxations, fractures, and tumors of the vertebræ, hemorrhage, inflammation, and tumors of the spinal membranes, and the like. Among the varieties of *toxic sciatic neuralgia* those occurring in the course of diabetes mellitus must be included, and which at times are bilateral and exceedingly obstinate. Bilateral and usually resistant sciatica is observed not rarely in cases of **tabes dorsalis**. The disease generally attacks *adults*, and *men* more commonly than women.

Anatomic Alterations.—But few reports have been made as to the anatomic alterations attending sciatica. In some cases the nerve is said to have been uninjured—*neurotic sciatica*—while in others it has been inflamed—*neuritic sciatica.* At times carcinomatous masses have penetrated the nerve from the neighborhood, and have developed within it. In a case of sciatica following purpura I found extensive hemorrhage into the nerve. At times collections of serous fluid have been reported as present in the nerve-sheath.

Symptoms and Diagnosis.—Sciatica is frequently recognized without difficulty, because the patient will indicate with his finger accurately the course of the sciatic nerve as the painful area. The pain extends from the point of exit of the sciatic nerve in the region of the hip down to the toes. Naturally, *partial sciatica* occurs at times, so that perhaps the pain extends only along the posterior aspect of the thigh, or along the calf of the leg, or is present only on the plantar aspect of the foot. Accordingly as the pain radiates from above downward or in the reverse direction a distinction has been made between *descending* and *ascending sciatica,* although this is not of much practical value, as many patients state that the pain occurs simultaneously throughout the whole course of the sciatic nerve. The number, duration, and severity of the attacks exhibit great variations. **Pressure-points** are present at the side of the sacrum, at the level of the posterior superior iliac spine, at the lower border of the gluteus corresponding to the point of exit of the sciatic nerve from the sciatic notch, at a point just behind the greater trochanter, at the middle of the posterior aspect of the thigh (bifurcation of the posterior cutaneous nerve of the thigh), in the popliteal space (tibial nerve), at a point just below the head of the fibula (peroneal nerve), and at a point each behind the external and the internal malleolus. **Vasomotor** and **trophic disturbances** are not common. Standing and walking cause increase in the severity of the pain, and for this reason the patient shields the neuralgic extremity. It is, therefore, not surprising that muscular **atrophy from disuse** slowly takes place. Rapid **degenerative muscular atrophy** occurs when the sciatica is of neuritic origin. Under such conditions **cutaneous anesthesia** and **impairment,** and even **abolition, of the knee-jerk** may readily be present.

On standing, it is not rarely observed that the spinal column undergoes lateral curvature—*scoliotic sciatica.* Generally the convexity of the curvature is directed toward the diseased side and less commonly toward the healthy side. An uncommon complication is the presence of *sugar in the urine.*

The *duration of sciatica* varies between a few weeks and many years. Some patients are never relieved of their disorder. Also, **relapses occur** readily and repeatedly. The danger of confounding **sciatica** and **coxitis** is particularly great. In the diagnosis of the latter the distinctive position of the lower extremity (flexion of the thigh and the leg with rotation outward) and the circumstance that kicking and rotatory movements of the thigh are painful in the presence of sciatica and usually are greatly interfered with, while they are free and unattended with pain in the presence of sciatica, must be taken into consideration.

Prognosis.—Sciatica is an obstinate and even an incurable disorder, but it is not attended with any immediate danger to life. Naturally, the patients may become greatly debilitated in consequence of pain and persistent insomnia, and also nervous.

Treatment.—In treatment an endeavor should first be made to meet the causal indications in the usual manner according to the suggestions already laid down. Under all circumstances **rest in bed** and an **equable temperature** are particularly serviceable measures. No specific remedy for sciatica is known, so that the treatment must be conducted upon the lines indicated on pp. 514–516. In chronic cases courses of **treatment with baths** have been resorted to, and indifferent thermal, saline, or sulphurous baths particularly have been recommended. At times I have secured successful results by means of **bloodless stretching of the sciatic nerve**, the extended extremity being flexed as strongly as possible at the hip-joint and moved toward the abdomen.

SPERMATIC NEURALGIA.

Spermatic neuralgia is attended with pain in the testicle and the epididymis, radiating in the course of the spermatic cord to the lumbar region. At times swelling of the testicle also occurs. Among the etiologic factors may be mentioned onanism, other sexual excesses, antecedent gonorrheal epididymitis, injuries, anemia, and neurasthenia. Further, excessive sexual continence has been thought to cause the disorder. The testicle should be supported with a suspensory bandage, narcotic ointments be applied by inunction, in the presence of severe pain an injection of morphin be made, electricity be employed, and the general condition be treated. In obstinate cases the painful testicle has been removed.

Neuralgia of the glans and the *penis*, the *scrotum*, the *labia majora*, the *urethra*, and the *perineum* has also been described. Among the causative factors are exposure to cold, injuries, and sexual excesses.

COCCYGODYNIA.

Neuralgia of the coccyx—coccygodynia—is attended with severe pain in the coccygeal region, which becomes increased to an intolerable degree on sitting, walking, and expulsive effort. The disorder generally occurs in women, and is attributed to injuries, difficulty in labor, or exposure to cold. Further, in many instances the condition appears to be really not a neuralgia, but an inflammatory condition of the coccyx. In obstinate and painful cases the coccyx has been removed.

ARTICULAR NEURALGIA.

Articular neuralgia occurs especially in pallid, nervous, or hysterical individuals. It may develop spontaneously or after exposure to cold or as a result of injury. Most commonly the knee-joint or the hip-joint is the seat of the disorder. In contradistinction from anatomically demonstrable arthritis, the patient usually keeps the extremity extended and avoids all movement. After the disorder has persisted for some time muscular atrophy from disuse may result. It is often exceedingly difficult to distinguish articular neuralgia from arthritis.

The **treatment** should be directed against the general condition. In addition, the patient should be encouraged to use the joint. At times it is necessary to insist upon such use of the joint. Occasionally recovery takes place abruptly.

ANESTHESIA.

Sensory disturbances of the skin and the mucous membranes are attended with either total or only partial loss of sensibility, and accordingly a distinction is made between **anesthesia** and **hyperesthesia**. Accordingly as all varieties of sensation or but a single variety is lost the result will be *total* or *partial anesthesia*, although the condition can be recognized only from a careful study of all varieties of sensation.

In what follows cutaneous anesthesia alone will be considered, although naturally conditions of anesthesia and hyperesthesia may occur wherever there are sensory nerves, as, for instance, in muscles, joints, fascia, tendons, periosteum, and internal viscera.

Cutaneous sensibility includes both the *sense of touch* and *common sensation*. Each of these principal divisions can be subdivided into several subgroups, and accordingly the following scheme of examination may be adopted:

Tactile Sensibility:
- Contact-sense;
- Pressure-sense;
- Localization-sense;
- Time-sense;
- Temperature-sense.

Common Sensibility:
Pain-sense;
Electric sensibility;
The sense of tickling.

In investigating cutaneous sensibility it should be a rule to employ the simplest *methods of examination.* In order not to distract the attention of the patient, it is well for him to keep his eyes closed during the examination, and if possible to assume a comfortable recumbent posture. *Contact-sensibility* is tested by touching or stroking the skin with a blunt body (the finger, a hair-brush, a bit of wood, a swab of cotton, a roll of paper). For purposes of comparison, symmetric portions of the skin or in the presence of para-anesthesia higher or lower portions should be tested successively. The test-agent should be neither too warm nor too cold, as the patient might confound the thermal sensation with that of contact. The simplest mode of testing the *pressure-sense* consists in making a varying degree of pressure upon the skin with the finger, or by placing upon the part to be tested a rather large coin at the temperature of the body and gradually adding more coins, and noting whether the patient is able to distinguish slight differences in weight. In making this examination the arms and the legs should be supported upon a firm base. The *sense of localization* consists in the ability of the patient to designate correctly the points upon the skin touched or to indicate them with his finger. In addition, it includes the recognition of the *area of contact.* The latter is determined by means of a pair of compasses or an esthesiometer (Figs. 73 and 74). In using either instrument, two movable points are applied simultaneously to the skin. The degree of separation of the points can be varied. The size of the tactile area consists in the smallest distance between the points at which the two are appreciated not as one, but as two. Examination of the *time-sense* is of little value. It is made by means of vibrating cords, on the sounding-board of which a healthy individual is capable of distinguishing the discontinuity of 1500 vibrations per second. An adequate method for testing the *temperature-sense* for practical purposes consists in blowing upon the skin and then breathing upon the skin, or by the application of test-tubes, one of which is gently warmed in the flame. *Paradoxic disturbances of the temperature-sense* are manifested at times in the confusion of hot with cold, and *vice versa.* For testing the *sense of pain,* needle-prick and the pulling of hairs are sufficient. The *electric sensibility of the skin* is tested by means of a dry metallic electrode connected with a faradic current, and the appreciation of the beginning prickling must be separated from the actual perception of pain. The *sensation of tickling* and of *itching* can be readily excited by touch with the finger upon the sole of the foot, the palm of the hand, in the axillary cavity, etc.

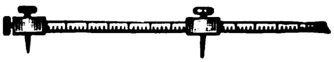


FIG. 74.—Esthesiometer.

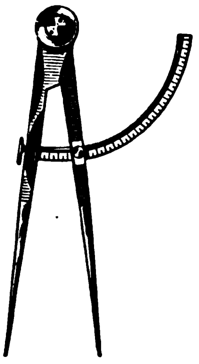


FIG. 73.—Compasses for testing sensibility.

Cutaneous anesthesia results from disease of either the peripheral receptive apparatus (sensory nerve-termination) or the peripheral sensory nerve-trunks, thus the conducting apparatus for sensory stimuli, or of the central receptive stations in the spinal cord or the brain, and it is by no means always easy to determine the seat of the disease with certainty. The demonstration of peripheral injuries to the skin itself or to the peripheral nerve-trunks is of particular importance in this connection. In addition, a study of the reflex processes is of great importance, as these are abolished in the presence of peripheral anesthesia, whereas they persist if the anesthesia is of central origin. As many nerve-trunks are of mixed character, it should not be surprising that often motor paralysis and anesthesia occur in association.

Etiology.—The *causes for cutaneous anesthesia* include the same agencies as give rise to paralysis and neuralgia, and, accordingly, a distinction can be made between *refrigeratory, traumatic, toxic,* and *infectious* cutaneous anesthesia, to which *vasomotor anesthesia* may be added. That cutaneous anesthesia may result from **exposure to intense cold** is indicated by the action of the ether-spray, which is employed upon the skin to render minor operations painless. The skin becomes anesthetic also when more considerable portions of the extremities are frozen. **Traumatic cutaneous anesthesia** occurs when a sensory nerve is compressed or contused. **Toxic cutaneous anesthesia** develops, for instance, when strong alkalies or acids are applied to the skin. Cutaneous anesthesia occurs also as a result of the action of lead and of carbon monoxid. Narcotics (morphin, cocain) may likewise diminish the sensibility of the skin. **Infectious cutaneous anesthesia** develops in the sequence of infectious diseases when inflammation of a sensory nerve has occurred.

Preservation of cutaneous sensibility depends further upon normal circulation of the blood, and the influence of cold, already mentioned, is probably due in part to resulting circulatory alterations. At times, however, such *vasomotor cutaneous anesthesia* develops spontaneously in consequence of spasm of the muscular coat of the vessels. Cutaneous anesthesia develops also after ligation of the extremities to the degree of pulselessness, and in conjunction with the occurrence of embolism or thrombosis of the main artery of an extremity and with thrombosis of the principal vein.

Cutaneous anesthesia can be readily demonstrated by means of the tests already described. The patient becomes conscious of the condition from his failure, in the presence of anesthesia in the hands, to feel a body grasped with these members, and which, therefore, he readily permits to fall, particularly if the guidance of the eyes is removed. Individuals with cutaneous anesthesia in the feet are not distinctly conscious of the resistance of the floor in walking. As a result, the gait may become uncertain and awkward. The patient walks with the feet held far apart, swinging them in walking, raising them unduly high, and he presents an ataxic gait—peripheral pseudotabes. Anesthesia of the skin and the trunk is attended with failure on the part of the patient to appreciate the presence of his clothing as well as the resistance of chairs or beds. Whether the condition is one of para-anesthesia, hemianesthesia, or anesthesia of an entire extremity or of only a single nerve, can be readily determined from the extent of the impairment of sensibility.

Symptoms.—At times there may be **paresthesia** in addition to cutaneous anesthesia, including a sense of tingling, of cold, and of stiffness. Some patients also complain of severe pain in the insensitive areas—**dolorose anesthesia.** This phenomenon may be explained by the fact that, for instance, a tumor by pressure may interrupt the conductivity of a sensory nerve, while, at the same time, the central termination of the nerve is irritated, and this irritation gives rise to pain, which, in accordance with the law of eccentric conduction, is referred to the peripheral distribution of the nerve. Often **motor, vasomotor,** and **trophic disorders** occur when cutaneous anesthesia is dependent upon disease of a mixed nerve.

The **prognosis** and the duration of the disorder vary with the nature of the causative condition.

In the **treatment** of cutaneous anesthesia, in addition to removal of the causative factors—**causal therapy**—cutaneous irritation plays an important part. This may be effected by means of *spirituous applications* (spirit of mustard, camphorated spirit, spirit of formic acid, compound spirit of angelica) or the *electric brush* of a faradic current or the kathode of a galvanic current.

TRIGEMINAL ANESTHESIA.

Etiology.—Peripheral trigeminal anesthesia occurs principally as a result of **exposure to cold** and of **injury.**

Punctured and incised wounds, neoplasms, and disease of the cranial bones are well adapted to cause trigeminal anesthesia. At times the condition is dependent upon abnormal conditions at the base of the skull (tumors, aneurysms, meningitic thickening), which exert compression upon and cause paralysis of the trunk of the trigeminal nerve. Under these conditions not individual, but all of the branches of the trigeminal nerve are involved in the anesthesia, and motor trigeminal paralysis (paralysis of the masticatory muscles) may be superadded.

Symptoms and Diagnosis.—Unilateral trigeminal anesthesia is attended with loss of **cutaneous sensibility** upon one side of the face and the anterior aspect of the auricle. When a glass or a spoon is carried to the mouth a sensation is perceived as if these objects were divided at the middle line, because one-half of the lip is insensible. Sensation is lost also upon the con-

junctiva, the **cornea,** and the **nasal and buccal mucous membrane.** Touching the eye causes no movement of the lid and no flow of tears through reflex influences. The secretion of tears is diminished. Tickling the nose upon the diseased side does not excite sneezing. Pungent olfactory impressions (ammonia, acetic acid) also are not appreciated. The sensibility of the buccal mucous membrane likewise is lost upon one side. The sense of taste is, as a rule, abolished upon the anterior two-thirds of the tongue. At times unilateral coating of the tongue and diminution in the secretion of saliva have been observed. The **facial expression** is frequently characterized by relaxation upon the diseased side, because the trigeminal nerve exercises a reflex influence upon the tone of the facial muscles. **Vasomotor** and particularly **trophic disorders** also occur. Among the latter inflammation of the cornea and of the whole eye—*neuroparalytic ophthalmia*—has long been known, although of late there is a growing tendency to adopt the view that this inflammation of the eye is not dependent upon the influence of trophic nerve-fibers, but that in consequence of deficient sensibility of the cornea winking is infrequent, so that foreign bodies from the air, and particularly bacteria, remain upon the eye and cause inflammation.

Peripheral is differentiated from **central trigeminal anesthesia** by the unaltered persistence of the **reflexes** with the latter.

Prognosis.—The prognosis depends upon the curability of the causative condition.

Treatment.—The treatment should be conducted according to the rules given on p. 528. In addition, the **eye should be protected** by means of a suitable bandage.

PERIPHERAL DISEASE OF THE NERVES OF SPECIAL SENSE.

DISEASE OF THE OLFACTORY NERVE.

Peripheral disease of the olfactory nerve is attended either with morbidly increased acuity of the sense of smell—*hyperosmia*—or with diminution and even loss thereof—*hyposmia* and *anosmia*, or with *parosmia;* that is, subjective appreciation of odors in the absence of odorous substances.

Hyperosmia is generally of central origin, and occurs with particular frequency in association with hysteria. Patients thus affected are capable of smelling substances that another with normal olfactory organs is incapable of appreciating; or they are greatly annoyed by olfactory impressions of not excessive intensity, and at times are seized with excitement, convulsions, or syncope.

Hyposmia and **anosmia** are common accompaniments of acute and chronic inflammation of the nasal mucous membrane. The condition has been frequently observed also in connection with nasal polypi. It often develops in the sequence of protracted ether-inhalation, as, for instance, in collectors of insects. It may occur as a result of traumatic influences in the presence of tumors of the ethmoid bone from pressure upon the fibers of the olfactory nerve or in the presence of fracture of the ethmoid bone or the skull. Tumors and meningitic thickening at the base of the skull, as well as tumors, hemorrhage, and softening in the frontal region of the brain close to the island of Reil, may cause anosmia through pressure upon or injury of the olfactory nerve. At times anosmia develops in advanced life, in consequence of involution of the olfactory bulb. Further, anosmia may be congenital in consequence of absence of the olfactory nerves. The condition may be unilateral or bilateral, and in many persons is detected purely by accident. Bilateral loss of the sense of smell is attended with disturbance in the sense of taste for all such articles of food and drink whose taste is dependent on their aroma.

Parosmia is frequently of central origin (mental disease, hysteria, epilepsy), although it may also occur as a result of peripheral influences, in connection with acute and chronic inflammation of the nasal mucous membrane. The condition generally found is one of a sense of disagreeable olfactory impressions (*cacosmia*), which not rarely gives rise to hallucinations in the insane. At times parosmia is associated with anosmia, a condition corresponding with painful anesthesia and susceptible of a similar explanation; that is, interruption of conduction on the one hand, and stimulation of the central extremity of the olfactory nerve on the other hand. Disturbances of the sense of smell can be tested with the aid of pleasantly smelling (oil of cloves, oil of rose) and disagreebly smelling substances (assafetida), as the disorder may be partial and not complete. Pungent odors (ammonia, acetic acid) should be avoided, as this variety of olfactory impressions is transmitted through the trigeminus. First one naris and then the other is tested, while the nasal orifice not being examined is carefully closed.

The **treatment** of all varieties of olfactory disturbance consists essentially in efforts to cure the fundamental disorder.

DISEASE OF THE GUSTATORY NERVE.

Disorders of the sense of taste on the anterior two-thirds of the tongue are dependent upon disease of either the trigeminal or the facial nerve, while those on the posterior third are dependent upon disease of the glossopharyngeal nerve. Further, it is also contended that the fibers for the sense of taste from the trigeminal nerve are derived from the trunk of the glossopharyngeal nerve. That the facial nerve itself contains no nerves of taste, but that these pass at intervals from the trunk of the trigeminal nerve into that of the facial nerve has already been mentioned on pp. 479 and 487 in the consideration of peripheral facial palsy. The morbid disorders of the sense of taste are manifested in a manner analogous to those of the sense of smell, and they are designated hypergeusia, hypogeusia, and parageusia.

Hypergeusia consists in an ability to detect the taste of substances even when present in small amounts. More pronounced gustatory impressions may give rise to conditions of excitement, convulsions, or syncope. Hypergeusia is generally of central origin, and is encountered with particular frequency in association with hysteria.

Hypogeusia and **ageusia** involving the anterior two-thirds of the tongue is most commonly unilateral in association with *peripheral facial palsy* if the lesion is situated between the geniculate ganglion and the giving off of the chorda tympani nerve. In other instances unilateral hypogeusia or ageusia is observed in association with trigeminal anesthesia or with injuries of the lingual nerve. Finally, the condition may involve the posterior third of the tongue in the presence of disease of the glossopharyngeal nerve. Persons with a heavily coated tongue often complain of impairment of the sense of taste, or this may develop after the ingestion of food that is unduly hot or irritating.

Parageusia is observed principally as a result of central causes, in the insane and the hysterical. The patient generally complains of a bad taste, although the mouth is empty.

The *tests for the sense of taste* are described on p. 483.

The **treatment** of disorders of the sense of taste is comprised in the relief of the primary disorder.

INFLAMMATORY AND DEGENERATIVE DISORDERS OF THE PERIPHERAL NERVES.

PRELIMINARY CONSIDERATIONS.

Inflammatory and degenerative disorders of the peripheral nerves are generally designated *neuritis*. It should, however, be borne in mind in this connection that inflammatory processes are by no means always operative, but it is difficult anatomically to make a sharp distinction between nerve-inflammation and nerve-degeneration. Nevertheless, neuritis plays a most important part at present in the domain of nervous diseases.

Etiology.—The *causes* for inflammatory and degenerative disorders of the peripheral nerves are **exposure to cold, traumatism, infection, and intoxication.** To what extent bacteria are concerned in these processes is as yet undetermined. It is highly probable that cold and injury act only as contributory influences for infection of peripheral nerves with bacteria, while in the development of infectious and toxic neuritis the injurious influence of toxins (bacterial poisons) or chemic poisons upon the nervous structures may be operative.

The **anatomic alterations** involve either the interstitial connective tissue of the nerves—*interstitial neuritis*—or the nerve-fibers themselves—*parenchymatous neuritis.* In addition, mixed varieties of the two principal classes mentioned occur—*mixed neuritis;* and these again may be separated into several subdivisions accordingly as the connective tissue and the nervous structure are affected simultaneously by the inflammatory irritant or the inflammatory process begins in one portion of the nerve and then involves the other secondarily.

Interstitial neuritis in its acute form is often indicated macroscopically by the thickened and unusually reddened appearance of the nerve, with loss of its transverse striation (bands of Fontana). Here and there extravasations of blood may have taken place, and these may be present both in the external nerve-sheath and upon transverse section of the nerve. The presence of numerous extravasations of blood has given rise to the designation *hemorrhagic* or *apoplectiform neuritis.* On *microscopic examination of the nerve* the blood-vessels are found greatly dilated, tortuous, and distended with blood. Their walls often appear thickened and swollen and glistening. The nuclei of the vessel-wall are in places increased in number and involved in fatty degeneration. In addition, the interstitial connective tissue is increased. At the same time it contains numerous cells and round cells.

In rare cases an accumulation of pus takes place in the nerve—*suppurative neuritis.* This may occur particularly when foreign bodies and toxic substances have penetrated into the nerve, and also when suppurative inflammation in the neighborhood of the nerve has extended to the latter.

In the presence of *chronic interstitial neuritis* the nerve frequently presents a brownish, almost black color, due to the remains of pigment from preceding hemorrhage. The nerve generally feels denser than normal, in correspondence with an increase in the interstitial connective tissue. At times the inflammatory areas appear as nodular enlargements—*nodose neuritis.* It is noteworthy that a neuritis by no means always extends throughout the entire course of a nerve, but involves only a single small portion or several portions, and accordingly a distinction should be made between *diffuse, circumscribed,* and *multiple* or *insular neuritis.* The neighborhood of joints and the points of deflection of nerves constitute especially favored situations for insular neuritis. At times a neuritis that is at first insular exhibits a tendency to extend progressively in the course of the nerve, and even to the spinal cord, and the condition is then designated *migratory neuritis.*

Parenchymatous neuritis can be diagnosed with certainty by means of microscopic examination. In teased preparations o fresh nerve-tissue or in such tissue stained with osmic acid fiber will be found whose medullary substance and axis-cylinder appea granular and involved in fatty degeneration, and often also absorbed, so that the empty nerve-sheath remains. In the latte multiplication of the nuclei takes place. On transverse section o hardened nerves empty sheaths will be found in greater or lesse number, and frequently containing but a single nucleus.

French physicians have pointed out that parenchymatous neuritis als at times involves the nerve-fibers for only short distances, and this condition has been designated *segmental neuritis*.

The **symptoms** of *neuritis* will naturally vary accordingly a the inflammatory process involves motor, sensory, or, as occur most commonly, mixed nerves. At the beginning of the diseas irritative symptoms are not rarely present, but these are shortl followed by paralytic phenomena. Therefore, *inflammation o motor nerves* is often at first attended with twitching and rigidit in the related muscles, but these are soon replaced by progressively increasing weakness and paralysis. The muscles rapidl undergo wasting, as they participate in the degenerative process and together with the diseased nerves are generally tender upo pressure. Both nerve and muscle exhibit degenerative electri reaction. In the distribution of the diseased nerves the tendon reflexes are at times increased at first, but generally diminutio and disappearance of reflex movement soon occur. After neuriti muscular paralysis has existed for some time muscular contractures and deformities readily develop.

Inflammation of sensory nerves is attended with especial frequency with **paresthesiæ**: a sense of coldness or the creeping o ants (formication), prickling and sticking of the skin, marke neuralgic pains. Often cutaneous hyperesthesia is present at th beginning of the disorder, but this subsequently is replaced b anesthesia.

In cases of *inflammation of mixed nerves*, in addition to th motor and sensory disturbances described, **vasomotor and trophi disturbances** are at times observed, as, for instance, pallor or cyanotic discoloration of the skin, accordingly as an irritative or paralytic condition of the vasomotor nerves is present, coldness o the skin, hyperidrosis or anidrosis, thickening of the epidermi abnormally increased desquamation of the epidermis, excessiv growth of hair, thickening and fragility of the nails, glossy finger, herpes or pemphigus-like cutaneous eruptions, articula swelling, etc.

The *course of a neuritis* may be acute, subacute, or chronic Acute neuritis at times sets in abruptly—*apoplectiform neuritis* In other instances it develops, like an acute infectious disease with rigor, fever, and enlargement of the spleen. Chronic neuritis may result from an acute neuritis or it may develop insidiously in an independent manner. At times neuritis begins first in a single nerve or in a few nerves, and then extends progressively to a larger number—*polyneuritis.* Under such circumstances it may happen that the alterations are first observed in the nerves of the lower extremity, gradually invading nerves at successively higher levels—*ascending neuritis.* Should cerebral nerves also become involved, the disease becomes a serious one, and death may readily take place in consequence of paralysis of the vagus-accessory nerve. Even when cerebral nerves are not involved neuritis is a serious disease in relation to its curability. While recovery is always possible, it is not certain, and under all circumstances much patience is necessary on the part of both patient and physician.

The **diagnosis** of *neuritis* is not particularly difficult. Inflammation of motor nerves is most likely to be confounded with **anterior poliomyelitis,** for in this disease also, as the lesion of the motor-trophic ganglion-cells in the anterior horns of the spinal cord is soon associated with degeneration of the related nerves and muscles, degenerative muscular atrophy and degenerative electric reaction occur; but poliomyelitis is unattended with sensory disturbances, and the diseased nerves and muscles are also usually free from pain on pressure. Neuritis is distinguished from other diseases of the spinal cord by the fact that the bladder and the rectum are almost always uninvolved with the former, while, on the other hand, nerve and muscle exhibit degenerative electric reaction.

The **treatment** of neuritis should be directed, in the first place, to the causative factors—**causal therapy.** In a case of refrigeratory neuritis, for instance, warm baths, salicylic acid, sodium salicylate, also diaphoretics (pilocarpin hydrochlorate) will be employed. Causal indications may be present also in a case of traumatic neuritis. Antecedent syphilis will require the employment of mercurial preparations and potassium iodid, and the like. The **symptomatic treatment** is the same as that in cases of peripheral paralysis (p. 475) and of cutaneous anesthesia (p. 528). Baths, salicylates, antipyrin, massage, and electricity are the most important remedies.

MULTIPLE NEURITIS.

Etiology.—Multiple neuritis, also known as polyneuritis, occurs at times like an *independent infectious disease,* and is then often referred, as a matter of convenience, to exposure to cold, the occurrence of which, however, cannot be confirmed on careful inquiry. At times cases of such infectious polyneuritis hr observed to occur in groups and in almost epidemic distribution. It must, nevertheless, be admitted that **exposure to cold** may be followed by multiple neuritis, at least in the sense that the exposure favors infection of peripheral nerves. At times multiple neuritis has been observed to develop in the sequence of **traumatism**, which at times has been of apparently insignificant character, as, for instance, riding upon rough roads. Multiple neuritis occurs with particular frequency in the sequence of **infectious diseases**. It has long been known and greatly feared in connection with pharyngeal diphtheria, but it may occur also in conjunction with typhoid fever, herpes zoster, dysentery, pneumonia, pulmonary tuberculosis, and particularly syphilis. Septic diseases, and particularly puerperal fever, at times give rise to multiple neuritis. A complete description of *toxic multiple neuritis* will not be attempted here, because it will be taken up in detail in succeeding sections. On the other hand, attention is to be directed to the occurrence of multiple neuritis in consequence of auto-intoxication. This condition is observed in connection with diabetes mellitus and gout, and possibly also cases of marasmus belong in this category, as, for instance, in association with carcinoma. With the exception of post-diphtheric paralysis, multiple neuritis is generally a disease of adults.

Anatomic Alterations.—Multiple neuritis may be of interstitial, parenchymatous, or mixed character, and nothing need be added to what has been stated on p. 532 concerning neuritis in general.

Symptoms and Diagnosis.—Multiple neuritis may pursue an *acute* or a *chronic* course. If it occurs as an independent *acute infectious disease*, it sets in at times with a chill, followed by elevation of temperature to as high as 40° C. (104° F.), and even higher. Often **enlargement of the spleen** is present. Above all, **paralysis** occurs, generally involving isolated nerves of the lower extremities at first, soon rendering the entire extremity helpless, and then extending to the upper extremities. Cerebral nerves also may be involved. Paralysis of the vago-accessory nerve is attended with great acceleration of pulse. Paralysis of deglutition may occur. At times paralysis of the hypoglossal and facial nerves has been observed. On one occasion I observed sudden blindness in consequence of inflammation of the optic nerve. By reason of the ascending character of the paralysis the disease is strongly suggestive of acute ascending spinal paralysis, but in cases of multiple neuritis the paralyzed nerves and muscles exhibit, in contradistinction from spinal paralysis, degenerative electric reaction. In addition, nerves and muscles are generally tender on pressure, and the paralyzed muscles soon undergo degenerative atrophy. Sensory disturbances are frequently present. **Vasomotor** and trophic disturbances (coldness and cyanotic discoloration of the skin, edema, articular swelling) also are observed. At times active delirium appears, and the patient may die in coma. The bladder and the rectum are, as a rule, uninvolved. In some instances I have observed recovery from acute multiple neuritis, although after a considerable time a second and even a third attack occurred—*recurrent polyneuritis.*

Chronic multiple neuritis is often unattended with fever, but in other instances periodic febrile movement occurs. The paralysis develops slowly in the course of weeks or months, and at times exhibits also an ascending character. The disease may be readily confounded with chronic anterior poliomyelitis, as in this disease also degenerative electric reaction is found in the paralyzed muscles, but it is unattended with sensory disturbances, and the nerves and muscles are not tender to touch. In some cases chronic multiple neuritis originates from an acute neuritis, the fever ceasing and the new paralysis developing but slowly. Long-continued paralysis is frequently attended with contractures that give rise to deformities in the extremities.

Prognosis.—The prognosis of multiple neuritis is always serious. If the disease pursues an ascending course, there will be danger of death from paralysis of cerebral nerves. In the presence of chronic multiple neuritis a long time may be expected to elapse before the diseased nerves will be regenerated and the paralysis will have disappeared.

Treatment.—The treatment of multiple neuritis should be conducted upon the lines laid down on p. 475.

TOXIC NEURITIS.

SATURNINE PARALYSIS.

Etiology.—Of the toxic varieties of neuritis, that due to lead-poisoning has been known longest. It occurs most commonly in certain artisans who in the pursuit of their avocation are exposed to chronic lead-poisoning. Among these are particularly painters, potters, printers, file-cutters, shot-molders, workmen in lead-mines, etc. Lead-poisoning may result accidentally from the use of preserved food kept in receptacles containing lead, of water transmitted through lead-pipes, of cosmetics containing lead, of snuff wrapped in lead-foil, etc. Rarely, lead-poisoning may result from carelessness on the part of the physician in consequence of the administration of preparations of lead in too large amounts or for too long a period.

Symptoms and Diagnosis.—The symptoms of saturnine paralysis usually appear only after the patient has been exposed to the action of the metal for a considerable length of time
a rule, other symptoms of lead-poisoning have alrea

ent, particularly the presence of a bluish-gray lead-line at the margin of the gums, together with anemia and colic. **Bilateral radial paralysis, with preservation of the function of the supinators,** is distinctive of lead-paralysis. The palsy generally develops gradually, and the patient complains at first of weakness in the hands and fingers, which eventually passes over into paralysis, and in spite of all devices renders the hand incapacitated for work. When the forearms are held horizontally the hands fall in palmar flexion, and at the same time the fingers are flexed and the thumbs adducted. Dorsal flexion of the hands, extension of the fingers, abduction and extension of the thumb, abduction and adduction of the hands toward the ulna or the radius, can be effected imperfectly, if at all. Of the extensors of the fingers, those of the index-finger and the little finger are involved late, so that both of these fingers can at first be relatively well engaged in dorsal flexion. Supination and extension of the forearm remain unaffected. Only rarely is lead-palsy widespread—*generalized lead-paralysis.* Under such circumstances the muscles of the upper arm, the shoulder, the back, and the lower extremities also may be involved. Even the muscles of the face and the larynx and the diaphragm are at times paralyzed.

The paralyzed muscles become the seat of **degenerative atrophy,** and both nerve and muscle exhibit **degenerative electric reaction.** The **tendon-reflexes** are abolished in the paralyzed parts, and gradually *muscular contractures* take place. Diseased nerves and muscles are generally tender on pressure. At times some of the tendons become thickened—*hypertrophic tenosynovitis.* **Paresthesia** and **hyperesthesia,** less commonly **anesthesia,** also occur.

The *duration of the paralysis* depends upon the degree of the disturbance, and more accurately upon the degenerative electric reaction. Advanced paralysis requires many months for recovery. Frequently *relapses* occur, often because the patients resume their former employment; less commonly relapses occur in spite of avoidance of contact with lead.

Anatomic Alterations.—Upon the basis of personal observation I should consider lead-paralysis the consequence of a *degenerative inflammation of peripheral nerves.* Paralysis of long standing may be complicated by secondary atrophy of the related trophic-motor ganglion-cells in the anterior horns of the spinal cord. The paralyzed muscles exhibit reduction in the size of their muscle-fibers, proliferation of the sarcolemma-nuclei, and increase in the interstitial connective tissue.

Prognosis.—Although lead-palsy is, as a rule, not a fatal disease, the prognosis with regard to permanent recovery is not favorable, because patients resume their injurious pursuits without observance of the necessary precautionary measures, or are even satisfied with an improvement in the paralysis and return to their work too quickly.

Treatment.—For the lead-paralysis itself **sulphur-baths** should be employed (potassium sulphid, 150.0—5 ounces—to a full bath at a temperature of 28° R.—35° C.—95° F.), **massage** of the paralyzed muscles be practised, or instead **electric treatment**, and thrice weekly subcutaneous injections of strychnin be made:

℞ Strychnin nitrate, 0.1 (1½ grains);
Glycerin,
Distilled water, each, 5.0 (75 minims).—M.
Dose: 0.25 (4 minims) subcutaneously.

Internally **potassium iodid** (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily) should be administered to eliminate the lead present in the system. As a **prophylactic measure** it is important that the patient shall never take a meal in the room in which lead is manipulated. Also, before eating, the mouth and the pharynx should be rinsed with water to remove lead-dust that may be present, and the hands should be freed from adherent particles of lead by washing. Experience has shown that drunkards are more susceptible to lead-poisoning than others.

In isolated cases paralysis has been observed among those exposed to the action of *mercury*, *zinc*, and *copper*, and this also is in all probability the result of a degenerative neuritis.

ARSENICAL PARALYSIS.

Arsenical paralysis is observed most commonly after *acute arsenical poisoning*. The extensor muscles of the leg, particularly the peronei, are generally first attacked, although the arms—with preference the extensors of the forearms—may also be involved. In one case I observed paralysis of the bladder and of the vagus. The paralyzed muscles rapidly undergo wasting and, together with the related nerves, are tender on pressure and yield degenerative electric reaction. The tendon-reflexes are abolished in the distribution of the paralysis. Gradually muscular contractures develop. Often the paralyzed parts become the seat of severe *neuralgia*. Nevertheless, cutaneous anesthesia becomes more and more marked, and as a result the gait becomes ataxic—*peripheral pseudotabes*.

Little is known with regard to the **anatomic alterations**, although the occurrence of a degenerative neuritis is most probable.

The **prognosis** is not unfavorable, although, naturally, many months may elapse before complete recovery takes place.

The **treatment** is the same as that for lead-paralysis.

Paralysis with symptoms similar to those dependent upon plumbic and arsenical poisoning has been observed also after poisoning with phosph
carbon monoxid, and carbon disulphid.

ALCOHOLIC PARALYSIS.

Etiology.—Alcoholic paralysis occurs principally in **drinkers of spirit.** In one instance I have observed the condition even in a boy.

Symptoms and Diagnosis.—The paralysis almost always develops gradually, although I have repeatedly observed a sudden (apoplectic) onset. Not rarely the paralysis has been preceded by severe **neuralgia,** which may also persist in association with the paralysis. The extensors of the legs and the forearms are involved in the paralysis earliest and most constantly. Gradually all of the muscles of the extremities may be affected, and even cerebral nerves, particularly the vagus, are at times involved. **Paralysis of the bladder and the rectum** is uncommon. The diseased nerves and muscles are tender on pressure, undergo wasting, and exhibit degenerative electric reaction. Contractures slowly develop. The tendon-reflexes are abolished in the paralyzed parts. **Cutaneous sensibility** is frequently impaired and **peripheral pseudotabes** may readily develop. At times marked swaying of the body occurs when the eyes are closed—**Bracht-Romberg's symptom.** Contraction of the pupil—**myosis**—and **sluggish reaction** to light may be present. **Paralysis of the ocular muscles** is uncommon. The disorder is at times attended with slight febrile movement. Progressive alcoholic marasmus, often associated with stupor, occasionally leads to a fatal termination. Two patients under my observation died in consequence of complicating miliary tuberculosis. The *duration of the disease* may be extended over many months.

Anatomic Alterations.—Degenerative atrophy of the nerve-fibers is found in the diseased nerves, and in the muscles atrophy of the muscle-fibers, increase in the sarcolemma-nuclei, and multiplication of the interstitial connective tissue. At times the degenerative process has extended from the nerves to the spinal cord, in which degenerative areas develop in the posterior columns and atrophy of the ganglion-cells in the anterior horns takes place.

Prognosis.—Recovery from extensive alcoholic paralysis is wholly possible, but requires a long time.

Treatment.—The treatment is the same as that for lead-paralysis (p. 538). Abstinence from alcohol is highly important.

At times paralysis has been observed after the ingestion of spoiled rye—*ergot-poisoning* or *ergotism*—and of spoiled maize—*pellagra*—and this must in all probability be considered the sequel of a toxic neuritis.

II. DISEASES OF THE SPINAL CORD.

PRELIMINARY DIAGNOSTIC CONSIDERATIONS.

In the diagnosis of disease of the spinal cord two points must be taken into consideration—in the first place, the *seat,* and, in the second, the *anatomic nature* of the spinal disorder. With regard to the seat of the disease a correct diagnosis is possible only from a knowledge of the *clinical anatomy and physiology of the spinal cord.* A brief *résumé* of these subjects may therefore be given. The spinal cord extends downward to the junction between the first and the second lumbar vertebra, whence it follows that all foci of disease below the second lumbar vertebra may involve the cauda equina, but not the spinal cord itself. Also, there is no danger from the introduction of instruments into the vertebral canal, as, for instance, in the practice of lumbar puncture, of inflicting injury upon the spinal cord, if this be undertaken below the second lumbar vertebra. Of the several segments of the spinal cord, the *cervical portion* is included between the first cervical and the second dorsal vertebra, and attains its greatest size at the level of the fifth and sixth cervical vertebræ. The *dorsal cord* is included between the second and the tenth dorsal vertebra, and the *lumbar cord* between the tenth dorsal and the second lumbar vertebra, its greatest size corresponding with the level of the twelfth dorsal vertebra.

In order to form a proper conception of the *vertebral column* it should be recalled that the spinous process of the seventh cervical vertebra projects markedly backward, whence the name *vertebra prominens,* and for which reason it can be readily recognized. If three contiguous spinous processes project conspicuously backward, the middle one may be considered as belonging to the seventh cervical. It is accordingly easy to localize accurately visible or palpable alterations in the vertebral column. Should, however, such external indications be absent, it will be necessary, in order to determine the *level of disease in the spinal cord,* to observe whether, on percussion of the vertebral column or on passing over it a sponge wrung out of hot water or by means of the kathode of a galvanic current, a circumscribed point of pain can be demonstrated; or, if cutaneous anesthesia be present, an attempt should be made to fix its upper limit at the side of the vertebral column. In the latter event, naturally, it should be borne in mind that the individual nerve-roots pursue a longer course in the vertebral canal, before they reach the intervertebral foramina and make their exit the lower the level at which they leave the spinal cord. fore, the upper limit of the anesthetic cutaneous area wil mately correspond with the level of the focus of disease only when this is situated in the upper portion of the spinal cord. The area of cutaneous anesthesia at the side of the spinal column will occupy a lower level than the seat of the disease if this be situated in the lower portion of the spinal cord. In general, the following rules will be found to hold good :

Foci of disease below the twelfth dorsal vertebra (*lower lumbar cord*) are attended with anesthesia in the lower extremities, except upon the outer and inner aspects of the thighs and the inner aspect of the legs (external cutaneous nerve of the thigh, obturator, crural, and saphenous nerves).

Foci of disease between the tenth and the eleventh dorsal vertebra (*upper lumbar cord*) are attended with anesthesia of the whole of the lower extremities.

In the presence of *foci of disease at the level of the eighth dorsal vertebra* (*point of exit for the tenth dorsal nerve*) the anesthesia extends to the level of the umbilicus.

Foci of disease between the fifth and the seventh dorsal vertebra (*points of exit for the sixth and seventh dorsal nerves*) give rise to anesthesia at the level of the ensiform cartilage.

Foci of disease at the level of the first dorsal vertebra give rise to anesthesia to the level of the nipples.

Foci of disease between the fourth and the fifth cervical vertebra give rise to anesthesia to the level of the clavicles.

Foci of disease at the level of the upper cervical vertebræ give rise to anesthesia in the arms.

For the comprehension of certain diseases of the spinal cord it is important to know that the *relation between the dura mater and the spinal column* differs from that between the dura mater and the bones of the skull, as in the cranium the dura is closely applied to the inner surface of the bones, while in the spinal canal it is separated from the inner surface of the vertebræ by loose connective tissue containing considerable fat and numerous veins. It therefore not rarely happens that foci of disease develop in the vertebral canal that may involve the spinal cord in consequence of pressure or by extension of inflammatory processes.

In *transverse sections of the spinal cord* two varieties of tissue may be distinguished by differences in color, an outer or *white substance* and an inner or *gray substance*. The white matter of the spinal cord consists principally of medullated nerve-fibers, and is rather a conducting organ, while the gray matter contains numerous ganglion-cells, and may be considered the seat of a series of spinal centers. In each half of the spinal cord an anterior and a posterior horn can be distinguished in the gray matter. The two sides of the gray matter are connected by means of the gray
[illegible] hape of the gray matter of the spinal cord has
[illegible] pared with the letter H, although it

will be readily discerned that the shape of the H varies in the different segments of the cord. It is well to train the eye to recognize from the shape of the gray matter the segment of the cord to which it belongs. In the lumbar segment the anterior and posterior horns are club-shaped (Fig. 75, 5, 6, 7), while in the dorsal region both horns are exceedingly small (Fig. 75, 4), and in the cervical portion the anterior horn particularly assumes a serrated appearance (Fig. 75, 1, 2, 3).

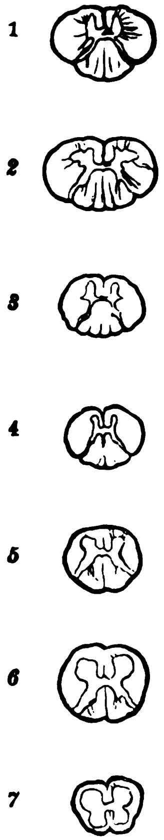


FIG. 75.—Transverse section of the spinal cord; natural size: 1, commencement, 2, middle, and 3, termination of the cervical enlargement; 4, middle of the dorsal cord; 5, commencement, 6, middle, and 7, termination of the lumbar enlargement (from personal preparations).

Both in the white and in the gray matter of the spinal cord definite, associated groups of nerve-fibers or of ganglion-cells can be distinguished, and these have been designated also *systems of the spinal cord*. They have been discovered in part by developmental methods, in part by experimental means, and from a study of diseases of the spinal cord.

In the *anterior horns of the spinal cord* the large multipolar ganglion-cells, which are generally arranged in three groups close together, form a connected system. Among their processes the axis-cylinder process in particular is of great importance, for soon after leaving the ganglion-cell it becomes surrounded by a medullary sheath and enters the anterior nerve-root of the spinal cord, then the peripheral motor nerve-trunk, and finally the related muscle-fiber. Accordingly, ganglion-cell, axis-cylinder process, peripheral nerve-fiber, and muscle constitute a continuous and uninterrupted whole, which has been designated a *neuron*, or, more accurately, a spinal-peripheral neuron or a neuron of the first degree.

Disease of the ganglion-cells of the anterior horns is known also as *anterior poliomyelitis* (*πολιός*, gray). The characteristics of such disease are relaxed (flaccid) paralysis of the related muscles, rapid degenerative muscular atrophy, degenerative electric reaction in the paralyzed nerves and muscles, abolition of the reflexes, preservation of cutaneous sensibility and of the muscular power of the bladder and the rectum. The ganglion-cells in question possess trophic-motor functions. If they are destroyed, the related axis-cylinder processes undergo degeneration, which extends to the muscle-fibers. Degenerated nerves and muscles yield degenerative electric reaction. These ganglion-cells do not possess sensory functions. The reflexes are abolished in the region controlled by the area of disease, because the transference of sensory stimuli to the motor paths within the spinal cord has been rendered impossible in consequence of the disease of the ganglion-cells.

In the gray matter of the dorsal portion of the spinal cord at the junction between the anterior and posterior horns there is an associated group of ganglion-cells bearing the name of the *column of Clarke* or the *vesicular column*. It is known that the fibers for the lateral cerebellar tract of the spinal cord, shortly to be mentioned, have their origin here, but nothing is known definitely with regard to their function.

The following columns or fiber-systems may be distinguished in the *white substance of the spinal cord* on either side:

1. The direct or anterior pyramidal tract,
2. The crossed or lateral pyramidal tract,
3. The lateral cerebellar tract,
4. Gowers' column or the antero-lateral fasciculus,
5. The postero-external or Burdach's column,
6. The postero-internal or Goll's column,
7. The antero-lateral remnant.

The *crossed or lateral pyramidal tracts* are situated in the lateral columns of the spinal cord, and are separated from the periphery of the cord in the cervical and dorsal regions by the lateral cerebellar tracts, while in the lumbar region they reach the periphery and at the same time assume a wedge-shaped appearance (Fig. 76, *2psb*). These tracts contain the largest portion of the motor fibers, which pass from the motor cortical centers in the anterior and posterior central convolutions, through the corona radiata, the internal capsule, the cerebral peduncle, the pons, and the medulla oblongata into the spinal cord, where they enter into relations with the motor-trophic ganglion-cells in the anterior horns, and from these conduct voluntary impulses to the periphery of the body. Each individual nerve-fiber of these tracts originates from the axis-cylinder process of a motor pyramidal cell, and continues without interruption to the lateral pyramidal tract of the spinal cord. Here the individual nerve-fiber passes into the gray matter of the spinal cord and breaks up into numerous ramifications. The latter come into contact with the ganglion-cells by means of similar ramifications (protoplasmic processes), which suffice for the transmission of voluntary impulses. The pyramidal cell, with its axis-cylinder process and the ramification of the latter in the gray matter of the spinal cord, is likewise designated a neuron, and it is described as a *neuron of the second degree* or a *cerebrospinal neuron*. Thus, the entire motor pyramidal (corticomuscular) tract, from the cerebral cortex to the muscle, is constituted of two neurons, which are connected within the anterior horn of the spinal cord by contact. The lateral pyramidal tract is known also as

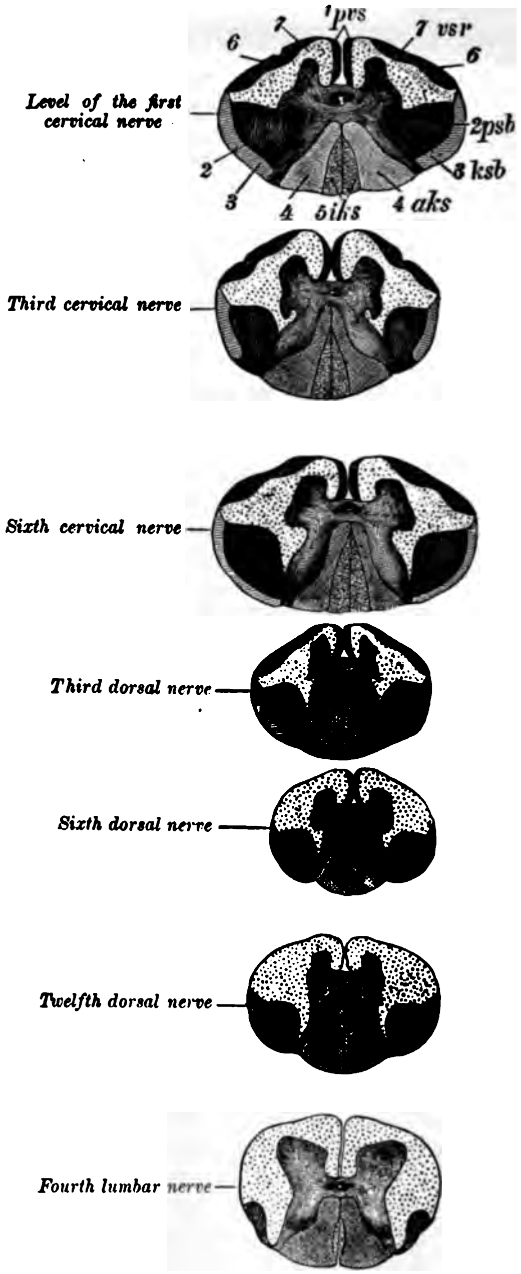


FIG. 76.—Diagrammatic representation of the principal columns of the spinal cord: *1pvs*, anterior pyramidal tracts (direct); *2psb*, lateral pyramidal tracts (crossed); *3ksb*, lateral cerebellar tracts; *4aks*, postero-external (Burdach's) wedge-shaped column; *5iks*, postero-internal (Goll's) column; *6*, Gowers' column; *7vsr*, anterolateral remnant, dividing into the ground-bundles of the anterior column and the remnant of the lateral column: magnified twice.

the *crossed* pyramidal tract because the motor fibers from the cerebral hemisphere upon one side largely pass over within the pyramidal decussation in the medulla oblongata to the lateral pyramidal tract of the opposite half of the spinal cord. From what has been said, it will therefore be understood that destruction of the pyramidal tract at any point in its course must be followed by motor paralysis. Should the disease be situated above the pyramidal decussation the paralysis will occur upon the opposite side of the body; but if, on the other hand, the disease is situated below the decussation, thus in the spinal cord itself, the paralysis will occur upon the same side. In contradistinction from paralysis resulting from anterior poliomyelitis, evidences of degenerative muscular atrophy and degenerative electric reaction are wanting in the paralyzed members; besides, the reflexes are preserved, because degeneration of the peripheral nerves and muscles and interruption of the spinal reflex arc do not occur, while, on the other hand, a tendency to spasm and muscular contracture is present, with exaggeration of the tendon-reflexes.

The *direct* or *anterior pyramidal tract* is situated in the anterior column of the cord, just beside the anterior longitudinal fissure (Fig. 76, *1pvs*). It is of subordinate importance and at times is wholly wanting. It is known as the *direct* pyramidal tract because it contains those portions of one corticomuscular pyramidal tract that do not cross over in the pyramidal decussation to the lateral pyramidal tract of the opposite half of the cord, but remain upon the same side, in order to enter the anterior pyramidal tract, and only within the spinal cord to pass over to the other side of the cord through the intermediation of the anterior spinal commissure.

Various differences occur in the *relation between the anterior and the lateral pyramidal tracts.* At times the anterior pyramidal tract is wholly wanting, or, as occurs less commonly, it preponderates over the lateral pyramidal tract.

The *lateral cerebellar tract* (Fig. 76, *3ksb*) is situated in the lateral columns of the cord close to the periphery, while internally it adjoins the lateral pyramidal tract. As has been mentioned, its fibers originate from the ganglion-cells of the columns of Clarke, and as the latter are present only in the dorsal cord, it will be understood that the lumbar cord contains no lateral cerebellar tract. The lateral cerebellar tract can be followed upward into the restiform body of the medulla oblongata, and thence into the superior vermiform process of the cerebellum. Coördinative functions are attributed to this tract, although this is based upon conjecture rather than upon fact.

A small portion of the white substance of the spinal cord situated posteriorly in the angle between the lateral cerebellar and the lateral pyramidal tract, and extending forward along the periphery of the cord as a narrow band to the neighborhood of the anterior longitudinal fissure, is known as the *column of Gowers* or the *anterolateral fasciculus*. Gowers believes this tract to conduct centripetally.

The *columns of Burdach and of Goll* in the posterior columns of the spinal cord are separated with particular distinctness in the cervical cord by a depression (posterior intermediate fissure). The columns of Goll contain fibers from the posterior spinal nerve-roots. The nerve-fibers that enter at the lowermost level are displaced progressively further forward and inward by those that enter at higher levels. Above, the fibers terminate in the nucleus funiculi gracilis, and they pass from here to the tegmentum, the quadrigeminate bodies, and the optic thalami. The nerve-fibers of the columns of Burdach reach the nucleus funiculi cuneati and the olive at the same level as the nucleus funiculi gracilis. As a unilateral lesion of the spinal cord gives rise to anesthesia upon the opposite side of the body, it must be concluded that the spinal sensory paths undergo decussation within the cord. This takes place in the posterior and perhaps also in the anterior spinal commissure.

The *anterolateral remnant* includes those nerve-fibers that are situated in the anterior and lateral columns of the spinal cord and do not belong to the pyramidal tracts, the lateral cerebellar tracts, and Gowers' tract (Fig. 76, 7*rsr*). They are considered to be short spinal paths whose function consists in connecting the ganglion-cells in the gray matter of the spinal cord with one another.

Disease of the spinal cord is revealed by *disturbances in the function of the cord*. The nature of these disturbances will depend upon the seat of the disease. Motor, sensory, vasomotor, secretory, trophic, and reflex disturbances may be present.

Spinal paralysis indicates disease either of the large ganglion-cells in the anterior horns of the spinal cord, or of the lateral pyramidal tracts. In each instance the character of the paralysis is different, as has been mentioned on pp. 542, 545.

Sensory disturbances will be present when the posterior columns of the cord or the posterior horns are diseased. It is believed that the fibers for painful and thermal sensibility pass through the horns, while the fibers for tactile sensibility pass through the posterior columns.

Vasomotor disturbances are not uncommonly associated with disease of the spinal cord. The vasomotor nerve-paths are believed to pass from the brain to the lateral column of the spinal cord, then to enter the anterior horn, and to extend from this through the anterior nerve-roots to the periphery of the body. In addition, there are *spinal vasomotor centers*, which are capable of acting independently of the brain, but nothing definite is known with regard to their situation.

Trophic disturbances occur, as has been mentioned, in muscles, fascia, bones, and joints in connection with anterior poliomyelitis. The posterior horns of the spinal cord also are credited with trophic functions, as trophic, that is, acute, gangrene of the skin, independent of pressure and infection, has been observed in connection with posterior poliomyelitis.

Alterations in the reflexes are manifested at times in exaggeration, at other times in abolition of reflex movement. The former occurs, for instance, in association with spastic spinal paralysis; the latter in association with tabes dorsalis. The spinal cord is necessary for the occurrence of the reflexes, because the transference of reflex irritation from the sensory to the motor paths takes place within the gray matter of the cord. Every interruption of the reflex arc will be attended with abolition of reflex movement, and this may naturally result from disease of the sensory or of the motor path or of the spinal cord. Exaggeration of reflex movement is often attributed to the withdrawal of the inhibitory activity of nerve-fibers entering the spinal cord from the brain.

The diseases of the spinal cord may be divided into two natural groups, namely: the *diseases of the structure of the cord* and *those of the spinal membranes*. The diseases of the spinal cord proper may be in turn irregular in distribution—*atypical* or *asystematic diseases of the spinal cord*; or they may remain confined to definite systems—*typical* or *systematic diseases of the spinal cord*. In the latter event a single system or several systems may be involved, and a distinction is accordingly made between *single* and *combined system-diseases of the spinal cord*. Diseases of the spinal cord in which it has not yet been possible to discover anatomic alterations are designated *functional diseases of the spinal cord*, or *neuroses of the spinal cord*.

DISEASES OF THE SPINAL CORD.

Atypical or Asystematic Diseases of the Spinal Cord.

ANEMIA OF THE SPINAL CORD.

The condition of anemia of the spinal cord is without clinical significance. Naturally, it will appear in the course of *general anemia*, such as may occur in consequence of excessive hemorrhage, in association with certain diseases of the blood (chlorosis, leukemia, pseudoleukemia, pernicious anemia), and following debilitating diseases (carcinoma, pulmonary tuberculosis, suppuration, chronic diarrhea). Such patients not rarely complain of spinal symptoms, such as paresthesia, hyperesthesia, or anesthesia, tremor, undue readiness of fatigue, and the like, symptoms that have been attributed to anemia of the spinal cord. Spinal anemia due to more especially local causes has been observed in connection with *embo-*

lism and *thrombosis of the abdominal aorta.* Under such circumstances there develop, in consequence of anemia of the lumbar cord, paraplegia and anesthesia in the lower extremities, paralysis of the bladder and the rectum, and abolition of reflex movement and electric irritability. Absence of pulsation in the femoral artery is distinctive of this condition. In consequence of an inadequate supply of blood gangrene of the lower extremities soon develops, and death occurs as the result of septicemia. In all cases of anemia of the spinal cord the **treatment** will consist principally in relief of the primary disorder.

HYPEREMIA OF THE SPINAL CORD.

Hyperemia of the spinal cord may result from increased arterial supply or obstructed venous exit, and a distinction is accordingly made between **arterial** and **venous hyperemia of the spinal cord.** Little of a definite nature is known concerning both conditions, and they are therefore as yet of no clinical significance.

Arterial hyperemia of the spinal cord may develop as a result of traumatism, infectious diseases, sexual excesses, and certain intoxications (alcohol, amyl nitrite); while *venous hyperemia of the spinal cord* develops in conjunction with stasis resulting from chronic disease of the heart, lungs, or liver, and convulsive disorders (tetanus, epilepsy, uremia).

Among the **symptoms** *of hyperemia of the spinal cord* a sense of traction in the sacral region, rigidity of the spinal column, girdle-sense, drawing and pain in the lower extremities, paresthesia, hyperesthesia, and anesthesia, and twitching and paralysis of the muscles have been mentioned. No characteristic symptom is known, and generally the diagnosis is made, in the presence of spinal symptoms in association with demonstrable causes of hyperemia of the spinal cord, by attributing the former to the latter.

The **treatment** should be directed less to the circulatory disturbances in the spinal cord than to the primary disorder.

HEMORRHAGE INTO THE SPINAL CORD.

Etiology.—Hemorrhage into the spinal cord or *hematomyelia* includes only such extravasations of blood as take place suddenly into the substance of a healthy spinal cord. Such hemorrhage is rare, and is to be carefully distinguished from the more common hemorrhage that takes place into inflamed and softened spinal tissue, and is designated *hematomyelitis.* Hemorrhage into the spinal cord may result from injury—*traumatic spinal hemorrhage.* The causative factors include not only concussion and injury of the vertebral column, but also heavy lifting, strong expulsive efforts, and the like. **Exposure to cold** is likewise not without influence; at least, a patient under my care was seized with hemorrhage into the spinal cord at a bathing-resort as a cold douche was directed against the vertebral column. Of importance are processes in the sexual sphere, particularly **venereal excesses** and **amenorrhea.** Suppression of **hemorrhoidal bleeding** also has been stated to provoke a tendency to spinal hemorrhage. Excessive indulgence in **alcohol** appears to be not without influence. Cases, however, occur also in which no exciting cause for the spinal hemorrhage can be demonstrated. The disorder develops most commonly in *men between the twentieth and the fortieth year of life.*

Anatomic Alterations.—Hemorrhage into the spinal cord may be classified according to the external appearance into transverse, longitudinal, and circumscribed. In cases of **transverse hemorrhage** into the spinal cord the extravasation involves the entire transverse section or it may be confined accidentally to one-half of the cord—**unilateral hemorrhage.** After incision of the dura the extravasation of blood can frequently be distinguished beneath the pia as a dark, reddish-black collection, which at times pushes the pia outward and projects above the level of the adjacent healthy spinal tissue. At times the pia and even the arachnoid are suffused with blood. Blood may also be admixed with the cerebrospinal fluid, and impart to this a reddish, bloody appearance. On section through the hemorrhagic focus the appearances vary with its age. In recent cases it consists of a soft bloody mass, constituted of red blood-corpuscles and disintegrated spinal tissue. The older the lesion the more conspicuous on microscopic examination are fatty degeneration and disorganization of the injured nerve-fibers. There are present numerous fatty granule-cells, which are nothing more than emigrated colorless blood-corpuscles, which have become laden with the not readily absorbed fat for the purpose of hastening its absorption. Older hemorrhagic foci are appreciable even to the unaided eye from their reddish-brown appearance, and they are likely to present a more firm consistence. Microscopically, in addition to fatty granule-cells, red blood-corpuscles are found in various stages of disorganization. Here and there granules, needles, and plates of precipitated hematoidin are also present.

Gradually a hemorrhagic extravasation may undergo complete absorption, and there will remain in its place a cavity filled with serous fluid whose wall is constituted by proliferated neuroglia—**apoplectic cyst.** In addition, **secondary degeneration of the spinal cord** occurs above and below the hemorrhagic extravasation: above, ascending degeneration of the columns of Goll, the lateral cerebellar tracts and Gowers' bundle; below, descending degeneration of the lateral and anterior pyramidal tracts. Only rarely will it be possible on microscopic examination to find the ruptured spinal vessel responsible for the hemorrhage. If the blood-vessels exhibit any change, this generally consists in secondary proliferation of nuclei and in fatty degeneration. Miliary aneurysms, which are as a rule responsible for hemorrhage into the brain, seldom occur in the spinal cord.

Tubular hemorrhage into the spinal cord extends especially in the longitudinal axis of the cord, and at times attains a length of several centimeters. Naturally, the white matter of the cord offers considerably more resistance to the extension of the hemorrhage than the gray matter. **Circumscribed hemorrhages** into the cord consist of small extravasations of blood that involve only certain small portions of the cord. The gray matter of the cord is more susceptible to all varieties of hemorrhage on account of its greater abundance of blood-vessels, and it is, therefore, a relatively common seat of circumscribed spinal hemorrhage. Small extravasations of blood in the spinal cord may disappear and leave only a brownish or yellowish cicatrix consisting of neuroglia—*apoplectic cicatrix*.

Symptoms and Diagnosis.—The **sudden occurrence** of spinal symptoms is distinctive of all varieties of hemorrhage into the spinal cord. In other respects the symptoms vary in accordance with the extent and the situation of the extravasation. *Transverse hemorrhage* in the lumbar or dorsal cord is attended with suddenly developed **paraplegia in the lower extremities.** At the same time there is **anesthesia.** The **tendon-reflexes,** particularly the knee-jerks, are generally enfeebled for the first few days, and this is thought to be due to the general concussion of the spinal cord induced by the hemorrhage; subsequently the tendon-reflexes become exaggerated. The tendon-reflexes remain permanently abolished only when the hemorrhage destroys the lumbar cord and thereby interrupts the spinal reflex arc. In the latter event **paralysis of the bladder and of the rectum** is present from the outset, as the spinal centers for the bladder and the rectum in the lowermost portion of the lumbar cord are destroyed; also in cases of transverse hemorrhage into the dorsal cord paralysis of the bladder and the rectum appears after the lapse of some time. The detrusor of the bladder suffers first, and the patients are capable of emptying the viscus only incompletely and with difficulty. Subsequently paralysis of the sphincter of the bladder is superadded, and the retention of urine at first present is succeeded by incontinence. Bacteria from the air and the urethra readily gain entrance into the bladder, and cause fermentation of the urine and readily excite cystitis, pyelonephritis, and *urinary septicemia.* Incontinence of urine also is attended with the danger of cutaneous irritation, of bed-sores, and of septicemia. In cases of transverse hemorrhage in the upper portion of the spinal cord symptoms of paraplegia of the arms occur in addition to the symptoms described. Hemorrhage high up may even give rise to bulbar symptoms if the nuclei of the cerebral nerves on the floor of the fourth ventricle or the intramedullary nerve-roots of individual cerebral nerves are involved in the morbid process.

With the occurrence of hemorrhage into the spinal cord the patients frequently complain of severe **pain** in the spinal column, which is probably dependent upon sudden distention of the pia mater by the extravasated blood. The **bodily temperature** remains unchanged, although at times slight elevation of temperature takes place in from three to five days, and this has been designated *reactionary fever*, and has been considered the sequel of an inflammatory reaction on the part of the spinal tissues surrounding the extravasation. If the transverse section of the spinal cord is wholly destroyed by the hemorrhage, recovery or even improvement cannot be anticipated, as regeneration of spinal tissue does not take place. Improvement can be hoped for only when part of the symptoms result from pressure exerted by the extravasated blood, and this pressure ceases after absorption of the blood has taken place.

After transverse hemorrhage into the spinal cord has existed for some time *muscular contractures* and *exaggeration of the tendon-reflexes* gradually develop in the paralyzed extremities, conditions that are commonly attributed to secondary degeneration of the lateral pyramidal tracts, although this has scarcely been demonstrated with any degree of certainty. The principal dangers from the disorder are bed-sores and septicemia and urinary decomposition, cystitis, pyelonephritis, and urinary septicemia.

Unilateral hemorrhage into the spinal cord sets in suddenly with the symptoms of a *unilateral lesion of the spinal cord*. Accordingly, paralysis is found upon the side of the hemorrhage and anesthesia upon the opposite side.

Circumscribed hemorrhage into the anterior horns of the spinal cord is attended with symptoms of anterior poliomyelitis (flaccid paralysis, degenerative muscular atrophy, degenerative electric reaction, abolition of the reflexes); and into the posterior horns, with partial anesthesia, particularly with loss of painful and of thermal sense.

Prognosis.—The prognosis of hemorrhage into the spinal cord is unfavorable, because such destruction of the cord as is effected is permanent. At times *gliosis* is said to have developed in the sequence of spinal hemorrhage.

Treatment.—For the control of hemorrhage into the spinal cord and to avert reactionary inflammation in the spinal tissue an **ice-bag** should be applied to the vertebral column at the site of hemorrhage. Success can scarcely be expected from the internal administration of **hemostatics**. To hasten absorption of the blood, sorbefacients, particularly potassium iodid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily), are often prescribed, but it is at least doubtful whether they have any effect. If symptoms of paralysis of the bladder and the rectum appear, attempts should be made in the presence of retention of urine to secure evacuation of the bladder thrice daily by means of compression of the bladder through the abdominal wall. The catheter should be resorted to only when the procedure mentioned proves unsuccessful, and in that event most rigid disinfection of the catheter must be secured. If incontinence of urine has developed, the urine should be collected in a suitable receptacle. The skin of the buttocks and the vicinity should be thoroughly cleansed morning and evening of all contamination, and washed with alcohol in order to avert **bed-sores.** It is, further, important to change the posture of the body several times during the day. If, in spite of these precautions, redness and discoloration of the skin appear, the surface should be covered with smoothly applied **adhesive plaster,** and the patient should be placed upon a hair mattress or a water-bed. If a bed-sore be present when the patient comes under observation, it should be covered with cotton that has been immersed in **camphorated wine,** or the patient should be placed permanently in a warm bath at a temperature of 28° R. (35° C.—95° F.). Not much will be accomplished with **electric treatment** of the spinal lesion and the paralyzed muscles. **Massage** of the paralyzed muscles should preferably be recommended in order to maintain the nutrition of the muscles and to avert the development of contractures.

ACUTE INFLAMMATION OF THE SPINAL CORD (ACUTE MYELITIS).

Etiology.—Acute inflammation of the spinal cord probably develops generally as a result of the activity of **bacteria,** and those influences that have hitherto been considered as the causes of the disease are probably without other significance than that of **contributory agencies for the infection.** Among these belong particularly *exposure to cold*, *traumatism*, and *preceding infectious disease.* Also, states of hyperemia of the spinal cord appear to favor infection of the cord. In women symptoms of spinal inflammation appear at times at the **menstrual period,** particularly if menstruation be absent or delayed. Myelitis is even reported to have been observed after profound **emotional disturbance**; for instance, after severe fright.

Inflammation of the spinal cord may arise by **extension from** disease of the vertebral column (carcinoma, tuberculosis). *Acute neuritis* also extends at times to the spinal cord. Acute myelitis is a rather uncommon disease, which attacks men somewhat more frequently than women, and which occurs particularly in adults.

Anatomic Alterations.—An acutely inflamed spinal cord is characterized by remarkable softness, so that the condition may be spoken of as *inflammatory spinal softening* or *inflammatory myelomalacia*. It should naturally be remembered that precisely the same alterations may take place if the circulation and nutrition of the spinal cord are cut off as a result of embolism or thrombosis of blood-vessels. There then occurs **necrotic spinal softening** or **necrotic myelomalacia**, although little of a definite nature is known with regard to this. In accordance with the duration of the disease the softened spinal cord presents a varying color, and accordingly a distinction has been made between *red*, *yellow*, and *gray softening of the spinal cord*. In the presence of red softening the color of the cord is due to the fulness of the vessels with blood and diapedesis of red blood-corpuscles. When numerous extravasations of blood occur the condition has been designated *[illegible]* or *hemorrhagic myelitis*. Destruction of migrated red blood-corpuscles, transformation of their hemoglobin, and progressive fatty degeneration of the nervous elements gradually give rise to the appearance of yellow softening of the spinal cord, which, when the hemoglobin is wholly absorbed, is succeeded by gray softening. The degree of softening is most variable. In some cases the inflamed spinal tissue presents an almost fluid consistence; [illegible] the same time it may happen that smaller foci of inflammation are gradually absorbed and in their place a **spinal cyst** [illegible] with serous fluid remains. Should the disease pursue a chronic course, induration or **sclerosis** of the previously softened tissue at times takes place. Rarely, cases of acute myelitis occur in which accumulations of pus take place in the spinal cord—**abscess of the spinal cord** (*purulent myelitis*).

On *microscopic* [illegible] fatty granule-[illegible] degenerated nerve-fibers [illegible] colorless red [illegible] and altered blood-[illegible] tion with [illegible] degeneration of [illegible] fatty granule-cells [illegible] Often the [illegible] corpuscles [illegible] and atrophy [illegible] marked [illegible] tinctness [illegible] increase and [illegible] ment. [illegible] takes place [illegible] oles [illegible] tures [illegible] for some [illegible]

Acute [illegible] substance

of the cord, perhaps because the large number of blood-vessels in this situation readily permits the entrance of numerous bacteria. Should the inflammation be confined to the gray matter of the spinal cord, the condition is spoken of as *central myelitis* or *poliomyelitis*. It is naturally the rule for the inflammatory process to extend from the gray to the white matter of the cord. Observations of inflammation of the white substance of the cord exclusively are rare, and are known as *leukomyelitis*. Should the inflammatory process involve especially the peripheral layers, the disease is designated *perimyelitis*. With this condition inflammation of the spinal meninges is frequently associated—*acute myelomeningitis*. The *distribution of acute myelitis* is extremely variable. At times it takes place principally throughout a transverse extent, so that the entire transverse section of the cord is involved in the inflammatory focus—*transverse myelitis*. An especial form of transverse myelitis consists in involvement of one-half of the spinal cord only. In some instances the inflammatory process exhibits a tendency rather to extend longitudinally, and at times it involves the entire length of the spinal cord—*diffuse myelitis*. Small myelitic areas, which are often recognized only on microscopic examination of the hardened spinal cord, are known as *circumscribed myelitis*, and if a number of such areas are present in the spinal cord separated from one another the condition is designated *disseminated* or *insular* or *multiple myelitis*. Experience has shown that the lumbar and dorsal portions of the cord are the most common seats of inflammation.

Symptoms and Diagnosis.—Acute myelitis not rarely begins, like an acute infectious disease, with a **chill**, followed by **fever**, with elevation of temperature to 39° or 40° C. (102.2° or 104° F.) and above. In some cases a sense of drawing in the back, a feeling of stiffness in the vertebral column, and paresthesiæ in the extremities appear as **prodromes**.

Symptoms of spinal paralysis soon appear, and these correspond most frequently with the clinical picture of transverse myelitis in the lumbar or dorsal cord. The patients complain of a sense of weakness in the lower extremities and are compelled to go to bed, and even within a short time flaccid paralysis of the extremities has developed. Painful jerking or muscular contractions also occur from time to time in the paralyzed parts. Complaint is frequently made of **paresthesiæ** in the paralyzed muscles, particularly the crawling of ants (formication), sticking, prickling, burning, and also sensations of cold.

e **anesthesia** is always more marked in the lower extremi-
The tendinous and cutaneous reflexes are wanting when the
portion of the lumbar cord is involved in the inflammatory
, and its functional activity is suppressed. When the in-
tory foci are situated at a higher level the reflexes remain

unchanged or are even unduly increased. Disease in the lower portion of the lumbar cord is from the first attended with *paralysis of the bladder and the rectum.* Of the muscles of the bladder, the detrusor is paralyzed first, and the patients are therefore unable to evacuate the urine, so that this fluid accumulates in the bladder, and may distend the latter to the level of the umbilicus, and even beyond that. Subsequently paralysis of the sphincter of the bladder is superadded, and the previous retention of urine is succeeded by incontinence with dribbling. Under such circumstances bacteria readily gain entrance from the air through the urethra into the bladder, where they cause alkaline decomposition of the urine, which becomes evident to the sense of smell as a disagreeably pungent urinous odor. Decomposed alkaline urine frequently gives rise to cystitis, pyelonephritis, and urinary septicemia, and the last is often a cause of death. In addition, it is scarcely possible to avoid wetting and irritation of the skin over the sacrum and the buttocks with decomposed urine, so that the development of **gangrene of the skin** or of a bed-sore is favored. This danger is further increased by the fact that in the presence of paralysis of the sphincter of the anus fecal matter also is voided involuntarily, and likewise causes irritation of the skin, and by the fact that if the patient lies constantly upon his back the weight of his body causes pressure upon the skin in the sacral region, with ischemia and a marked tendency to the development of gangrene and inflammatory alterations in marked degree. Some patients complain of a constricting or even a painful **girdle-sense** about the trunk, which is generally attributed to irritation of the posterior spinal nerve-roots. Pressure upon and percussion of the vertebral column at times induce pain.

Occasionally **vasomotor, secretory,** and **trophic disturbances** are observed. The paralyzed members appear unusually pale or strikingly red, and accordingly feel cool or warm. Not rarely excessive secretion of sweat takes place in the lower extremities or the paralyzed members remain dry in the presence of generalized sweating. *Edema* also develops, and is generally attributable to stagnation in the lymphatic circulation in consequence of deficient activity. Among the trophic disturbances some forms of *bed-sore* have been included, which develop with unusual rapidity in situations that are exposed neither to pressure nor to want of cleanliness.

Acute myelitis may exhibit a *stationary* or a *progressive character.* In the former event the symptoms are not progressive, while in the latter they exhibit an unmistakable tendency to extend more and more. Under these circumstances the condition is usually attended with the alterations of *acute ascending myelitis.* The paralysis begins in the lower extremities, extends then to the abdominal and subsequently to the thoracic muscles, in the course of a few days to the arms, and finally also to the cerebral nerves.

There occur respiratory disturbances, difficulty in swallowing, acceleration and frequently also irregularity in the action of the heart, and finally death from suffocation or paralysis of the heart. Paralysis of the phrenic nerve and the diaphragm also readily causes death by suffocation. If pains be taken to determine daily the upper limit of cutaneous anesthesia, it will be easily possible to follow accurately the extension of the inflammatory process in the spinal cord.

Among the *complications* of acute myelitis, cystitis and *urinary septicemia*, and bed-sores and general septicemia, are by far the most common and the most serious. Bed-sores form with especial frequency over the sacrum, next in frequency over the trochanters, the malleoli, and the heels. They often extend in a frightful manner in extent and depth, and the sacrum may as a result be destroyed and the vertebral column opened. Acute myelitis may terminate fatally within a few days, particularly if it assume a rapidly ascending character; or the initial fever gradually subsides, but after the lapse of several weeks death occurs as a result of septicemia or of urinary infection; or the disease assumes a chronic course and persists for months or years. Under the conditions last named a portion of the paralytic phenomena may disappear in so far as these are dependent rather upon compression than upon actual destruction of nervous elements. Complete recovery is scarcely to be expected. Further, the disease exhibits a great tendency to *relapse*, in consequence of insignificant injurious influences.

The **diagnosis** of acute transverse and of progressive myelitis is not difficult. In contradistinction from **hemorrhage into the spinal cord**, importance should be attached to the fact that the symptoms do not set in suddenly, but successively. In the presence of acute **spinal meningitis** irritative symptoms predominate, particularly pain. The presence of rigidity of the neck also is of importance. **Spinal ascending (Landry's) paralysis** is unattended with sensory disturbances, as well as paralysis of the bladder and the rectum. **Multiple neuritis** is attended with degenerative muscular atrophy and degenerative electric reaction in the paralyzed nerves and muscles, while the bladder and the rectum usually remain unaffected. From the clinical course described such cases of acute myelitis will naturally deviate in which the inflammatory process is confined to one-half of the spinal cord or to small circumscribed areas. In the first event, the symptoms of a unilateral lesion of the spinal cord appear acutely (paralysis and hyperesthesia upon the side of the lesion, and anesthesia upon the opposite side), while in the latter they depend upon the varying seat of the inflammatory focus.

Prognosis.—The prognosis of acute myelitis is grave, as there is always immediate danger (urinary infection, septicemia, paralysis of cerebral nerves), or the disease pursues a chronic course; complete recovery can scarcely be expected.

Treatment.—In the presence of acute myelitis **an ice-bag,** or, still better, the **ice-sac of Chapman,** should be applied to the vertebral column. For the control of the fever, and thereby to diminish the flow of blood to the spinal cord, **sodium salicylate** or **phenacetin** should be prescribed:

℞ Sodium salicylate,	1.0 (15 grains).
Make 10 such starch-capsules.	
Dose: 1 powder every two hours.	

℞ Phenacetin,	1.0 (15 grains);
Sugar,	0.5 (7½ ").—M.
Make 10 such powders.	
Dose: 1 powder thrice daily.	

The **diet** should be exclusively liquid, and a milk-diet is the best. Strong coffee or tea and wine and alcoholics should be forbidden. Daily **evacuation of the bowels** should be secured, and to this end some stewed fruit (apple-sauce, prunes) should be given at the midday meal. If necessary, laxatives should be prescribed, and glycerin-enemata are particularly to be recommended. It is important for the **position** of the patient to be changed every two hours, in order to avoid persistent pressure upon the same part of the skin. Care should also be taken to have the sheet always free from creases and that it does not contain dry bed-crumbs. The entire body should be cleansed with tepid water at least once daily, and the back and the sacral region should be rubbed with some form of spirit, cologne-water, or lemon-juice, in order to increase the resistance of the skin. If inflammation of the skin and bed-sores threaten in spite of these measures, the skin should be covered with smoothly applied adhesive plaster, and the patient should be placed upon an air-mattress or a water-bed. The greatest consideration should be given to such paralysis of the bladder and the rectum as may be present. If retention of urine occur, an effort should first be made to evacuate the bladder by pressure through the abdominal walls, and only if this fails should resort be had to the use of the catheter, which must be most scrupulously disinfected. If dribbling of urine be present, a proper receptacle should be provided. If the skin is soiled by urine or feces, it should at once be cleansed and washed with a solution of mercuric chlorid (1.0 : 1000). If a bed-sore be present when the patient comes under observation, it should be dressed with cotton that has been immersed in spirit of camphor or the patient should be placed in a permanent tepid bath (30° C.—24° R.—86° F.).

The foregoing directions for the care of the patient are of far greater importance than any medicament. It is at least questionable whether **antiphlogistics** (mercurials, leeches, cups), **sorbefacients** (**potassium** iodid), or nervines (bromids, belladonna, ergot, arsenic, preparations of gold or silver, strychnin, etc.) are capable of exerting any effect. Also the utility of **electric treatment** of the spinal cord and the paralyzed muscles is doubtful. On the other hand, resort is generally had to **massage** of the paralyzed muscles, in order to maintain their nutrition and avert the development of contractures.

CHRONIC INFLAMMATION OF THE SPINAL CORD (CHRONIC MYELITIS).

Etiology.—Chronic myelitis is dependent upon the same causes as acute myelitis, and accordingly a distinction has been made principally between **refrigeratory, traumatic,** and **infectious chronic myelitis.** Among the infectious varieties syphilitic myelitis should be particularly mentioned. Without doubt, alcoholism also plays an etiologic role, and under such circumstances the myelitis would be considered **toxic.**

Anatomic Alterations.—Spinal tissue in a state of chronic inflammation is characterized generally by increased hardness or sclerosis. Not alone do the diseased areas feel unusually hard, but they also appear gray, translucent, and shrunken. It is noteworthy that the spinal cord may appear unaltered in the fresh state, although after hardening in preparations of chromic acid, as, for instance, Müller's fluid, the inflammatory areas can be recognized readily from their light-yellow color.

On *microscopic examination* hyperplasia of the neuroglia and destruction of nerve-fibers and ganglion-cells will be found in the inflamed areas. Of the nerve-fibers, the medullary sheaths first undergo granular disintegration and fatty degeneration. The disappearance of the axis-cylinders is often preceded by marked swelling. The ganglion-cells undergo contraction to round and often greatly pigmented structures, without processes. The blood-vessels appear dilated in places, are distended with blood, exhibit striated and thickened walls with many nuclei, and are surrounded on their outer surface by collections of round cells. The adventitial lymph-spaces also not rarely contain round cells. Fatty granules are encountered only in small number, in contradistinction from acute myelitis, while amyloid bodies are present in considerably greater number.

Chronic myelitis exhibits the same distribution as acute myelitis. Disease of the gray matter is known as *chronic central myelitis* or *chronic poliomyelitis,* while disease of the white matter is designated *leukomyelitis.* Should the chronic inflammatory process remain confined to the peripheral layers of the white matter of the spinal cord, the condition is designated also *chronic perimyelitis.* Under the conditions last named the spinal membranes are frequently involved in the inflammatory process, and particularly the pia mater and the arachnoid undergo thickening and adhesion—*chronic myelomeningitis.* For the same reasons that are applicable to acute myelitis a distinction can be made between *chronic transverse myelitis, unilateral myelitis, circumscribed, diffuse,* and *chronic myelitis.*

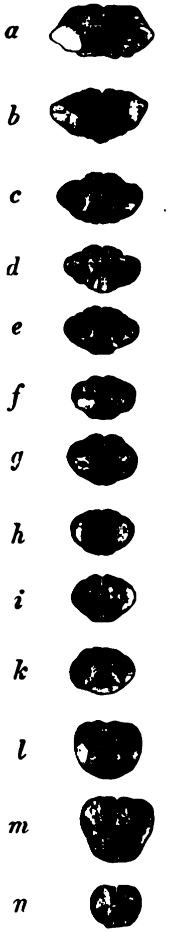
Symptoms, Diagnosis, and Prognosis.—When chronic myelitis occurs as an independent disorder it generally develops gradually. Pains in the course of the vertebral column, a sense of constriction, shooting pains in the arms and the legs, paresthesiæ in the lower extremities, and at times also vesical disturbances are often complained of as prodromes. Most commonly chronic myelitis is situated in the lumbar or dorsal cord, and in conformity with this fact the more serious disturbances generally appear first in the lower extremities. The legs become readily fatigued in walking, the knees often give way, and the patient stumbles over slight irregularities or elevations upon the floor. Fatigue and weakness in the lower extremities progress gradually in such a way that the patient shortly resorts to the use of crutches for support. Eventually complete **paralysis** ensues, and the patient is kept permanently in bed or in a chair. To the motor paralysis **sensory disturbances** are generally superadded, most commonly more or less marked and complete anesthesia. **Vasomotor** and **trophic alterations** may also be present. The **tendinous** and **cutaneous reflexes** are abolished when the inflammation has interrupted the functional activity of the lumbar cord. Exaggeration of the reflexes occurs in connection with transverse myelitis of the dorsal cord when secondary degeneration of the lateral pyramidal tracts takes place below the inflammatory focus. Then muscular contractures generally develop, most commonly in the flexors of the thigh and the leg. The **bladder** and the **rectum** frequently exhibit disturbances. At times retention and incontinence of urine alternate. Some patients complain of **priapism** or of **impotence**. Chronic myelitis is generally unattended with fever. At times transitory febrile movement occurs, during which the inflammatory process advances. At times it extends from below upward—*chronic ascending myelitis*. To the paralysis of the lower extremities there is then superadded paralysis of the arms, and should the process extend to the medulla oblongata and the pons Varolii there will develop paralysis of various cerebral nerves (hypoglossal, vago-accessory), which may lead to a fatal termination in consequence of paralysis of deglutition or of the heart.

The *duration of the disease* may extend over many years. Recovery is possible in cases that are not too far advanced. Generally, however, it is not complete. The principal dangers are cystitis and urinary infection or bed-sores and septicemia.

Treatment.—The treatment of chronic myelitis is the same as that of acute myelitis. The well-to-do may, in addition, undertake courses of treatment at the **baths** during the summer. There may be mentioned indifferent baths (Gastein, Pfäffers, Ragatz, Wildbad), saline baths (Nauheim, Rehme, Kissingen, Reichenhall, Rheinfelden, Bex), sulphur-baths (Aachen, Baden in Aargau, Baden near Vienna, Schinzach, Stachelberg, Leuk, Lenk, Serneus), and **peat-baths** (Marienbad, Franzensbad, Elster, Cudowa, Steben).

MULTIPLE CEREBROSPINAL SCLEROSIS.

Etiology.—Little of a reliable nature is known with regard the causes of multiple cerebrospinal sclerosis. **Exposure to l, traumatism, antecedent infectious disease, and intoxications** (alcohol, lead, phosphorus, carbon monoxid) are considered causes of the disorder. At times the disease has occurred as a sequel of the **puerperium.** In some cases **heredity** has been observed; in others, several members of a family have been attacked—*familial form.* The disorder generally develops between the fifteenth and the thirty-fifth year of life. It occurs but rarely in childhood, although isolated cases of *congenital multiple sclerosis* have been reported.



IG. 77.—Transverse sec-
ıs of the spinal cord from
ase of multiple cerebro-
ıal sclerosis, in a man 35
rs old: *a–d*, cervical por-
ı: *f–k*, dorsal portion;
lumbar portion; natural
: hardened in Muller's
d (personal observation,
ich clinic).

Anatomic Alterations.—Multiple cerebrospinal sclerosis is characterized by the formation of **foci of neuroglia,** which are distributed throughout the entire central nervous system in varying size, number, and arrangement. The white nervous matter everywhere appears particularly favored by the foci (Fig. 77). The individual foci can be readily recognized, for when of sufficient size they are sharply differentiated from the surrounding healthy structure by their reddish-gray or grayish color. Generally they feel indurated, whence the term sclerosis. Often they can be detected through the pia mater, so that the brain and the spinal cord present a grayish, mottled appearance. On transverse section of the spinal cord they frequently become depressed. The size of the foci may attain several centimeters; but, on the other hand, it may be so inconsiderable that the smallest foci can be recognized only with the aid of a microscope. Often they are present side by side in large number and above one another, while in other instances they occur in but small number. Their distribution follows no rule, and no case resembles another. In the medulla oblongata sclerotic areas are not rarely found in the nuclei of individual cerebral nerves. Similar changes have been observed also

Microscopic examination discloses *proliferation of the neuroglia* and destruction of the nerve-fibers in the sclerotic areas. It is noteworthy that the axis-cylinder offers resistance for a long time, so that *naked axis-cylinders* may be found in the areas. The *blood-vessels* further exhibit alterations, which consist in thickening of their wall, nuclear proliferation, fatty degeneration and the presence of round cells, fatty granule-cells, and also yellowish flakes of pigment in the adventitial lymph-sheaths. The cells in the sclerotic foci have been thought to consist, in part, of emigrated colorless blood-corpuscles. *Fatty granule-cells*, and particularly *amyloid bodies*, are frequently encountered in the sclerotic areas. In all probability multiple cerebrospinal sclerosis must be considered as a form of *disseminated chronic myelitis*, the inflammatory irritant being brought to the spinal cord through the blood-vessels.

Symptoms.—The distinctive symptoms of multiple cerebrospinal sclerosis are generally preceded by **prodromes** of an indefinite character. These include vertigo, headache, paresthesiæ, neuralgia, disturbances of the bladder, and paroxysmal vomiting (gastric crises). The disease can be recognized only when **typical symptoms** make their appearance, including intention-tremor, scanning speech, nystagmus, and apoplectiform attacks. *Intention-tremor* may be recognized from the fact that when the patients wish to execute a movement the members set in action begin to tremble actively or generally to shake, and in greater degree the more nearly they approach the consummation of the act. If the patient attempts to touch with his fingers a body held before him, the arms and hands are thrown to and fro. The introduction of a needle into a previously made perforation is scarcely possible, because the fingers and the needle are too actively moved to and fro upon the paper. On attempts to carry a glass or a spoon to the mouth the contents are partially spilt by the shaking of the arm, and the rattling of the teeth can be heard. Writing, sewing, dressing, buttoning are naturally disturbed in marked degree, and they may even be rendered impossible. On sitting erect, in walking, and in the use of a stick the oscillating and tremulous movements may be readily observed.

Opinions are divided as to the *mode of origin of intention-tremor*. It has been thought that the naked axis-cylinders in the sclerotic areas are capable of transmitting the impulses of the will only interruptedly. According to others, transverse radiation is believed to be possible from non-medullated nerve-fibers, and thus to disturb the normal transmission of volitional impulses. It is less probable that intention-tremor is dependent upon the presence of sclerotic areas in definite portions of the brain, as the phenomenon is a most constant and early manifestation, so that it would be necessary to assume the presence invariably of early disease of definite portions of the brain, whereas the disorder is characterized especially by the irregular distribution of the foci of disease.

Scanning speech is characterized, in the first place, by the articulation of the individual syllables of a word in jerks and with pauses, and, besides, by the peculiar, monotonous, singing, and high-pitched character of the voice. The disturbances of speech are thought to be dependent upon intention-tremor of the muscles of articulation and of the larynx or of the vocal bands. Not rarely most marked distortion of the face is observed on attempts to speak. A patient under my care was capable of speaking only by holding the lower jaw firmly with her hands, and keeping it at rest. Sudden dilatation of the chink of the glottis and tremulous movements of the vocal bands during phonation have also been observed on laryngoscopic examination.

The *nystagmus* is likewise due to intention-tremor of the ocular muscles. It can be readily recognized from the horizontal to-and-fro movement of the eyes that takes place when a finger is moved to and fro in a horizontal direction, and which becomes the more marked the further the finger is moved outward. Rarely, twitching movements in a vertical direction or rotatory movements occur—*vertical* and *rotatory nystagmus*. Nystagmus occurs rather less constantly than intention-tremor and scanning speech, and is present in about one-half of the cases.

Apoplectic attacks occur still less commonly. Consciousness is lost, and when it is restored the patient finds himself paralyzed upon one side of the body or in one member; but, as a rule, the paralysis disappears in the course of a few days. There is a disposition to attribute such attacks, which not rarely are attended with elevation of temperature and are at times accompanied also by eclamptic convulsions, to the development of new sclerotic areas.

The typical symptoms of multiple cerebrospinal sclerosis must be distinguished from the more *accidental nervous disturbances*, which vary accordingly as one or another portion of the central nervous system is the seat of the sclerotic process, and is thus thrown out of function. As of the anatomic alterations, it may likewise be said of this group of symptoms that one case scarcely resembles wholly any other.

Of diagnostic significance are particularly **atrophic changes in the optic nerve,** which preferably involve the temporal half of the optic papilla. At times disturbances occur also in other cerebral nerves (paralysis of the ocular muscles, of the trigeminus, of the vagus, tinnitus aurium, impairment of hearing, immobility of the tongue, etc.). Frequently **motor disturbances** appear, particularly paresis or paralysis in the lower extremities. These are often associated with muscular spasm and exaggeration of the tendon-reflexes. The gait becomes spastic-paretic, and the clinical picture resembles that of so-called spastic spinal paralysis. **Sensory disturbances,** generally partial anesthesia, are also not uncommon occurrences. At times *paralysis of the bladder* develops, which may subside, subsequently, however, to reappear.

If groups of ganglion-cells in the anterior horns of the spinal cord are destroyed by the sclerotic process, **muscular paralysis with degenerative atrophy** and degenerative electric **reaction may** result. When paralysis has existed for some time **muscular contractures** may develop, frequently earliest in the adductors of the thighs, subsequently in the flexors of the thighs, the legs, and the feet. Extensive foci in the vicinity of the posterior columns of the spinal cord render the **gait** ataxic, and under such conditions confusion with tabes dorsalis may arise.

Vasomotor and **trophic disturbances** have been observed, but are among the less common manifestations. The **mental condition** not rarely suffers. The patients become more and more apathetic, forgetful, and finally demented. Sometimes uncontrollable laughter is observed at an early stage, long-continued laughter of an unusually hearty, almost boisterous character being induced on slight provocation. Uncontrollable crying also may occur.

The *course* of multiple cerebrospinal sclerosis frequently extends over many years, and often remissions and exacerbations in the symptoms alternate. Death results from accidental disease (pulmonary tuberculosis, pneumonia, etc.), or from excessive exhaustion, or from urinary infection in the presence of paralysis of the bladder, or from septicemia in consequence of bed-sores, or from bulbar paralysis (paralysis of swallowing and of respiration).

Diagnosis.—The diagnosis of multiple cerebrospinal sclerosis is easy in typical cases. Naturally, emphasis must be placed upon the fact that there is a **neurosis of the central nervous system**—so-called *pseudosclerosis*—which may exactly reproduce the clinical picture of multiple sclerosis, although upon post-mortem examination the central nervous system may be found unchanged. The occurrence of atrophy of the optic nerve is distinctive of cerebrospinal sclerosis.

Intention-tremor occurs at times also in association with cerebral tumors, mercurial poisoning, and hysteria; but under such conditions the remaining symptoms of multiple sclerosis will be wanting.

Should the typical symptoms of the disease already described be absent, it will scarcely be possible to make the diagnosis with certainty. The disease may then be readily confounded with chronic myelitis, tabes dorsalis, spastic spinal paralysis, and amyotrophic lateral sclerosis. Confusion with paretic dementia is also possible. Further, it should be borne in mind that no invariable relation exists between the severity of the morbid manifestations and the extent of the anatomic alterations.

Prognosis.—Multiple cerebrospinal sclerosis is an incurable disease, and from this point of view the prognosis is unfavorable. It is true that at times such remarkable improvement occurs as to encourage the hope of recovery, but this proves deceptive. There is, however, no immediate danger of death.

Treatment.—No specific remedy for multiple sclerosis is known, and, in general, the rules should be followed that have been laid down on p. 485 for the treatment of myelitis.

TUMORS OF THE SPINAL CORD.

Tumors of the spinal cord are not uncommon: **gliomata, sarcomata, carcinomata, myxomata, cholesteatomata, gummata, and tuberculomata** have been observed.

Anatomic Alterations.—Tumors of the spinal cord are generally of spherical form, but at times they involve the entire transverse section or one-half or a still smaller section of the spinal cord, or they may extend throughout the length of the cord from above downward. At times they are clearly defined from the surrounding spinal tissue by a connective-tissue capsule, but at other times they gradually merge into the structure of the cord. Often the latter is softened at the point of junction. In the tumors themselves softening and the formation of cavities may also take place. In tumors well supplied with blood-vessels extensive hemorrhage may suddenly occur, with destruction of the neoplastic tissue and of the adjacent tissue of the cord.

The **causes** for tumors of the spinal cord are often unknown. At times traumatism, pregnancy, and the puerperium are named as causes. At times the tumors are of metastatic origin. Gummata and tuberculomata are sequels of syphilis and tuberculosis respectively.

The **diagnosis** of a spinal tumor is scarcely ever possible. This is a matter of course for cases in which spinal symptoms are wholly wanting. The same condition also prevails at times in connection with relatively extensive neoplasms if these displace rather than destroy the nervous elements of the cord. Under other circumstances, in accordance with the seat and the extent of the new-growth, symptoms of transverse interruption or of a unilateral lesion of the cord, of anterior poliomyelitis, syringomyelia, spastic spinal paralysis, tabes dorsalis, and the like, may be present, and only the autopsy may reveal the causes for the spinal disturbances.

The **prognosis** is serious. Death results generally from progressive exhaustion, in the presence of paralysis of the bladder from urinary infection and in the presence of bed-sores from septicemia.

Specific **treatment** is applicable only when syphilis is present, but otherwise the treatment applicable to myelitis should be employed (p. 557).

CAVITIES IN THE SPINAL CORD (SYRINGOMYELIA AND HYDROMYELIA).

Anatomic Alterations.—The presence of cavities in the spinal cord is at times due to morbid dilatation of the central canal, and the condition is then designated *hydr* [illegible] It is characterized by the presence in places of cylindric epithelium upon the walls of the cavity. The disorder has been observed particularly in connection with tumors in the posterior fossa of the skull, and it has been attributed to stasis. Most cavities in the spinal cord result from *gliosis* (*gliomatosis*); that is, proliferation of neuroglia first takes place, which not rarely extends throughout the entire length of the spinal cord, and even through the medulla oblongata, and then by the softening of this tissue a cavity results—*syringomyelia*. Less commonly cavities result from softening of myelitic foci or of tumors of the spinal cord.

Hydromyelia and syringomyelia are by no means always easily and with certainty distinguishable from each other, for when syringomyelia is present rupture into the central canal occasionally takes place, and there may be subsequent dilatation of the central canal, and the walls of the cavity then likewise present cylindric epithelium in places. The cavity in a case of syringomyelia may involve the entire spinal cord and also the medulla oblongata, and it may terminate in the fourth ventricle, or it may develop for only a short distance at a single point or at several points in the spinal cord. Most commonly the cervical cord is the seat of the disease, and in this situation the cavity usually attains its greatest size. In the course of the spinal cord the cavity often alters its position (Fig. 78). At times it is situated between the posterior columns of the spinal cord, while at other times it involves the gray matter of the cord, now in the neighborhood of the anterior horns, where it uses destruction of the large mo- ic ganglion-cells, then in the neighborhood of the posterior

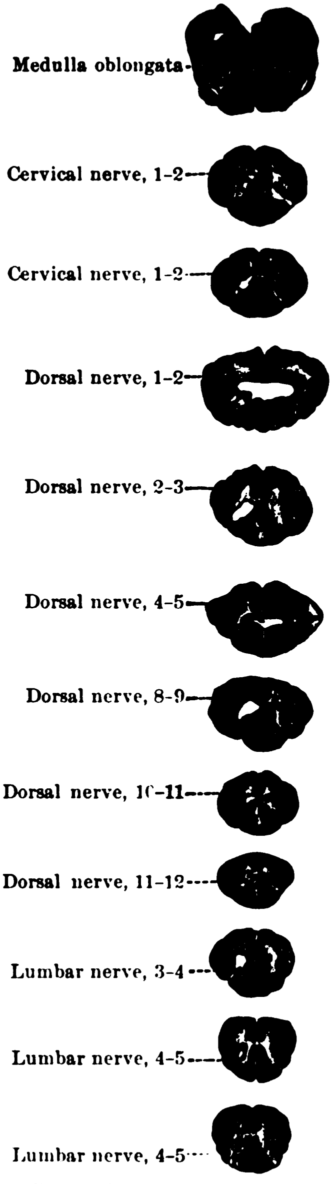


FIG. 78.—Syringomyelia in a woman 29 years old; natural size (personal observation).

horns, in other situations in the middle portion of the gray matter, etc. The size of the cavity varies from the scarcely visible to such an extent that the tip of the little finger can be readily introduced. Under the latter condition the entire transverse section of the cord is increased in size. The spinal cord not rarely resembles a fluctuating sac oscillating to and fro, and which on incision gives exit to a generally clear and but rarely flocculent or blood-stained fluid, and which collapses after evacuation. The cavity is generally provided with a membrane formed of dense neuroglia. The blood-vessels are generally thickened and obstructed by thrombi, conditions that are associated with the softening of the neuroglia and the formation of the cavity. Often yellowish and brownish pigment is encountered in the neighborhood of the cavity. The adjacent columns of the spinal cord are not rarely sclerotic.

Etiology.—Syringomyelia is probably in many cases dependent upon **developmental defects** in the spinal cord that undergo further development as a result of **accidental influences.** The latter include particularly *traumatism* and *antecedent infectious disease.* **Hereditary** and **familial occurrence** of syringomyelia has also been observed. The condition generally appears in *adults.*

Symptoms.—An extensive cavity in the spinal cord may be wholly unattended with symptoms. Among the distinctive manifestations of syringomyelia are **progressive muscular atrophy, partial loss of sensibility,** and **vasomotor** and **trophic changes.**

Progressive muscular atrophy generally begins, like typical spinal muscular atrophy, in the muscles of the thenar and hypothenar eminences and in the interosseous muscles. Next, the extensor muscles upon the dorsal aspect of the forearm and the deltoid are involved. The muscles waste progressively, become correspondingly weaker, exhibit fascicular (fibrillary) contractions and degenerative electric reaction. The condition can scarcely be differentiated from chronic anterior poliomyelitis, upon which spinal progressive muscular atrophy depends, if partial anesthesia also is not present. The development of the symptoms is readily explained by the fact that the cavity has destroyed the large motor-trophic ganglion-cells in the anterior horns of the spinal cord.

Partial anesthesia occurs in cases of syringomyelia together with or independently of progressive muscular atrophy, and is due to destruction of the posterior horns of the spinal cord, through which pass the paths for painful and thermal sensibility. It is not surprising, therefore, that loss of *painful* and *thermal sensibility* occurs. Needle-pricks are often appreciated only as touch. Often the skin is marked by the wounds of burns, which are frequently acquired from contact with a stove, the attention of the patients not being attracted to their wounds by the pain, but by the odor of burning tissue. At times cold is felt as warm, and *vice versa.*

Among the *trophic disorders* ulceration, particularly on the fingers, should be mentioned, occurring and persisting without pain. At times also the terminal phalanges are exfoliated without pain. Often the phalanges become peculiarly shortened and thickened. Not rarely contracture of the aponeurosis of the hand is observed. Marked kyphoscoliosis in the upper dorsal portion of the spinal column frequently develops. At times painless fissures form upon the fingers. Bullæ also often form, suggestive of herpes or pemphigus. At times a perforating ulcer develops. Spontaneous fracture of bones and alterations in joints are also known to occur.

Among *vasomotor manifestations* erythema, edema of the hands or feet, anidrosis and hyperidrosis may be mentioned. Not rarely *inequality of the pupils* exists. *Bulbar symptoms* also develop when the cavity has invaded the medulla oblongata. Among the symptoms may be mentioned atrophy of the tongue, unilateral facial atrophy, paralysis of deglutition, paralysis of the muscles of the vocal bands, accelerated action of the heart, glycosuria, and polyuria. At times paralysis of the ocular muscles develops.

Optic neuritis and contraction of the visual field also have been observed in a number of instances. At times the patients have exhibited ataxia or a spastic-paretic gait.

The *course of syringomyelia* is chronic. Death results from accidental diseases, progressive marasmus, bulbar paralysis, bedsores, or septicemia.

Diagnosis.—The recognition of syringomyelia is easy in typical cases. Some of the symptoms are suggestive of **nervous leprosy**, but this disease occurs only in the tropics, and can be recognized bacterioscopically from the presence of leprosy-bacilli. The symptom-complex described by Morvan, and designated *Morvan's disease*, and which is characterized especially by inflammation and painless exfoliation of the terminal phalanges, is only syringomyelia.

Prognosis.—Syringomyelia is an incurable disease, and its prognosis is therefore unfavorable. Life may, however, be maintained for many years.

Treatment.—The treatment is limited to the relief of particularly troublesome or dangerous symptoms.

SPINAL COMPRESSION-PARALYSIS.

Etiology.—Spinal pressure-paralysis results most commonly in consequence of **disease of the spinal column.** Tuberculosis of the vertebræ particularly, carcinoma of the vertebræ less commonly, give rise to spinal pressure-paralysis. The condition may result also from luxation or fracture of the vertebræ, from arthritic or syphilitic exostoses, from thickening of the odontoid process, and the like. At times processes that exert pressure upon the spinal cord enter the vertebral canal from without; for instance, cheesy-tuberculous inflammation extending from the pleural cavity through the intervertebral foramina into the spinal canal; carcinomatous changes in the kidney, which have followed the same path in their extension; aortic aneurysm, first perforating the vertebral column and then compressing the spinal cord; and disintegrating carcinoma of the esophagus, which injures the spinal cord after destruction of the vertebral column. Pressure upon the spinal cord sometimes arises from the *spinal membranes*, and occurs particularly in connection with meningeal hemorrhage, inflammation, and neoplasm. *Disease of the spinal cord* itself is also easily capable of exerting injurious pressure upon the diseased organ, particularly progressively growing tumors of the spinal cord. Spinal pressure-paralysis may occur at any *period of life*. Thus, it is not rarely induced in children by tuberculosis of the vertebræ.

Anatomic Alterations.—Spinal pressure-paralysis is frequently attended with **alterations in the vertebral column**, particularly kyphosis (gibbus). Under such conditions it may be inferred that stenosis of the vertebral canal is responsible for the compression of the spinal cord, although such an occurrence is but seldom actually encountered. In the majority of cases the inflammation of the vertebræ or in the vicinity gives rise to inflammation upon the outer surface of the spinal dura mater—external spinal pachymeningitis—and the pressure upon the spinal cord results as a secondary consequence. In the presence of tuberculosis of the diseased vertebræ it is the rule, for instance, that in the vicinity of the diseased vertebræ thick, cheesy-tuberculous masses are present upon the outer surface of the dura, and these represent the actual cause of the compression. The effects of the alterations named upon the **spinal cord** itself are variable. In some cases the spinal cord appears compressed, diminished in size, marked by a deep constriction, and not rarely hardened or sclerotic, although these occurrences are rather infrequent. More commonly either the spinal cord exhibits no alteration in size at the point of compression or it is widened, and on touch is found unusually soft. On transverse section it rises above the level of the cut surface, appears of grayish or reddish-gray color, and its cut surface fails to present the usual appearance or presents it but imperfectly. Also, after the spinal cord has been hardened in Müller's fluid the confused appearance of the cut surface is visible, and even more distinctly than in the fresh state. Besides, it will be found, if the disease has existed for a considerable length of time, that **second-**
y degeneration of the anterior and lateral pyramidal tracts below
d of the columns of Goll, the lateral cerebellar tracts, and
wers' bundle above the point of compression has taken place.

Microscopic examination **discloses degeneration of nerve-fibers at the point of compression. Often naked axis-cylinders, whose medullary sheaths have been destroyed by granular disintegration and fatty degeneration, are conspicuous for their marked swelling. The neuroglia appears increased and generally contains numerous fatty granule-cells. The blood-vessels are often greatly distended and markedly tortuous. Here and there collections of round cells are present in the neighborhood of the blood-vessels. In the gray matter of the spinal cord the ganglion-cells undergo swelling at first, and frequently present vacuoles; subsequently they shrink into small structures without processes. It was formerly believed that all of these symptoms were inflammatory, and therefore the designation *compression-myelitis* has also been employed; but this is inappropriate, because the alterations are often in the main the results of anemic states and venous stasis, which necessarily are followed by the alterations in the meninges.**

Symptoms and Diagnosis.—The symptoms of spinal pressure-paralysis are frequently preceded by **prodromal** local alterations in the vertebral column, particularly circumscribed spinal pain, appearing spontaneously or upon pressure, and distortion of the spinal column, and at times also burrowing abscesses. Patients also often complain of a constricting girdle or hoop-like sensation about the trunk, which is induced by irritation of posterior nerve-roots. The distinctive symptoms of compression of the spinal cord consist in signs of **transverse interruption of the functions of the cord.** At first these are incomplete, and involve the motor paths alone or principally, whilst subsequently sensory disturbances also become apparent. The predominance of motor symptoms is explained by the fact that pressure-paralysis of the spinal cord is often associated with disease of the vertebral column, and this generally arises from the bodies of the vertebræ, which are in close proximity to the motor paths of the spinal cord. Besides, as has been determined by experiments upon animals, the motor nerves offer less resistance to pressure than the sensory nerves.

Frequently the clinical picture begins with weakness in the lower extremities, which more or less quickly becomes transformed into complete *paralysis* and compels the patients to remain in bed. The paralysis is at first flaccid. If the focus of pressure is situated in the upper portion of the cervical cord, the arms in addition to the legs will be paralyzed. The *tendon-reflexes*, as well as the *cutaneous reflexes*, are generally increased. Often *vesical disturbances* are present, consisting at first in retention of urine (detrusor-paralysis), and subsequently in incontinence of urine (sphincter-paralysis). In consequence there may arise the danger of alkaline decomposition of the urine, cystitis, pyelonephritis, urinary infection, bed-sores, and septicemia. *Paralysis of the rectum* also occurs frequently, and increases the danger of bed-sores and septicemia. The patients often complain of *muscular twitching* in the paralyzed members, and this may occur spontaneously or result from reflex irritation on palpation of the extremities, and is at times attended with severe pain. Not rarely severe shooting *pain* occurs paroxysmally in the paralyzed extremities. *Paresthesiæ* (formication, tingling, prickling, burning, a sense of cold) also occur. *Cutaneous anesthesia,* in conjunction with paralysis, indicates almost total transverse interruption of the spinal cord. At first it is often incomplete, subsequently, however, becoming complete. Should demonstrable alterations in the vertebral column be absent, it will be important to determine the upper limit of anesthesia in order to localize the seat of pressure. *Vasomotor* and *trophic disturbances* (abnormal color of the skin, anidrosis, hyperidrosis, the formation of blisters) are not rarely observed.

The *course of the disease* is generally chronic; either there is a tendency to progressive aggravation, or periods of improvement and exacerbation frequently alternate with each other. Recovery is not impossible when the focus of pressure disappears and the compression of the spinal cord itself has not given rise to alterations in the substance of the cord of too serious a nature. When paralysis has existed for a considerable length of time, *muscular contractures* often develop, at first generally in the extensors, but subsequently in the flexors of the thighs and the legs.

The *diagnosis of a transverse lesion of the spinal cord* is in itself generally easy, but insurmountable difficulties may arise in endeavoring to attribute the disturbance to spinal compression. In this connection the fact whether local alterations in the vertebral column (painful pressure-points, but especially deformities of the spinal column) are present or not, is particularly important. Should these be absent the diagnosis will remain doubtful. The determination of the nature of the compressing influence is also frequently attended with difficulty. In early life pressure-paralysis of apparently spontaneous origin, with a projection of the vertebral column, generally depends upon tuberculosis of the vertebræ. The presence of painful anesthesia is to a certain degree distinctive of carcinoma of the vertebræ. The patient under such circumstances complains of the most intense pain in the extremities, whose cutaneous covering is totally insensitive. The condition is explained by the fact that the pressure of the new-growth upon the posterior spinal nerve-roots has interrupted the transmission of sensory impulses from the periphery to the brain, although irritation of the central terminations of the nerve-roots induces severe pain that is referred to the periphery. In order to determine the cause of pressure-paralysis all the organs in proximity to the vertebral column should be examined most carefully.

Prognosis.—The prognosis of spinal pressure-paralysis is unfavorable under all conditions. Progressive exhaustion, paralysis of the bladder and urinary infection, bed-sores and septicæmia, are the principal dangers. Some of the causes of compression (carcinoma, aneurysm of the aorta) render the prognosis unfavorable by reason of their incurable nature.

Treatment.—**Causal treatment** should be employed when the pressure is of syphilitic origin, and it should consist in inunctions of mercurial ointment and the internal administration of potassium iodid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily). **Rest** is necessary under all circumstances. In addition, an **ice-bag** should be applied continuously over the focus of disease, and **massage** of the paralyzed members should be practised. Internally, *potassium iodid* may be essayed. Recently the vertebral column has been opened in several instances, and the compressing focus of disease removed.

UNILATERAL LESIONS OF THE SPINAL CORD.

Etiology.—A unilateral lesion of the spinal cord is one that interrupts the functions of one-half of the cord. It may be caused by a **punctured wound** of the spinal cord, although it may develop also in connection with **inflammation, hemorrhage, new-growths, and compression** of the cord.

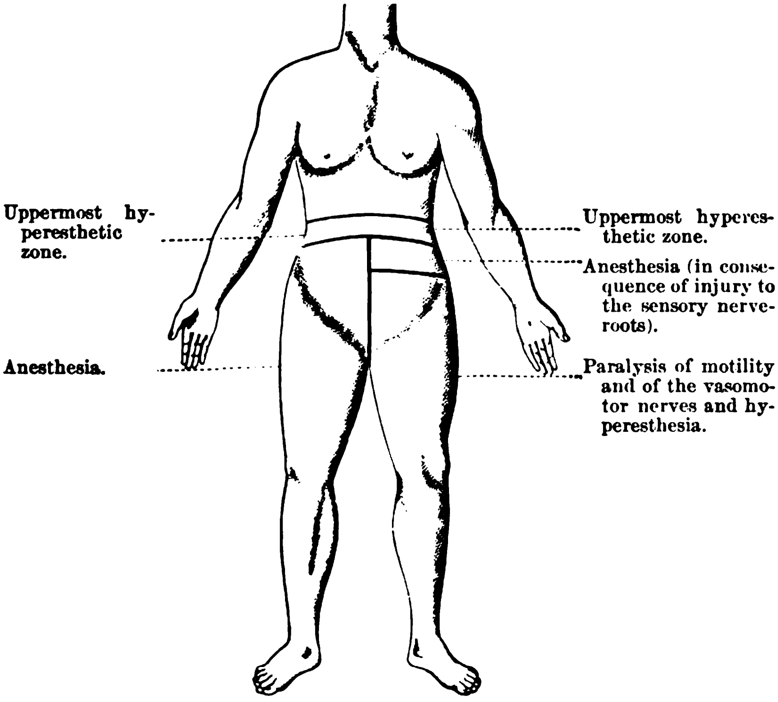

Uppermost hyperesthetic zone.
Anesthesia.
Uppermost hyperesthetic zone.
Anesthesia (in consequence of injury to the sensory nerve-roots).
Paralysis of motility and of the vasomotor nerves and hyperesthesia.

FIG. 79.—Diagram illustrating the symptoms of a unilateral lesion of the left half of the dorsal cord.

Symptoms and Diagnosis.—Unilateral lesions of the spinal cord give rise to striking and readily recognized symptoms, namely, paralysis and cutaneous hyperesthesia upon the side of the injury and anesthesia upon the opposite side. Accordingly, **spinal hemiplegia** and **crossed anesthesia** are distinctive of a unilateral lesion of the spinal cord. Upon the paralyzed side the muscular sense, motor power, and electromuscular sensibility are diminished. Vasomotor and at times trophic disturbances also are observed. Reflex irritability is unaltered or increased. If the cutaneous hyperesthesia is traced upward, a narrow band of cutaneous anesthesia will be detected surrounding the trunk upon the paralyzed side from the middle line to the vertebral column, and referable to injury of posterior nerve-roots. Above this band is a hyperesthetic zone surrounding the trunk entirely (Fig. 79). The distribution of the phenomena described depends upon the seat of the disease, and at times is confined to the lower extremities, while at other times it involves also the arms. In the presence of disease of the cervical cord **symptoms of paralysis of the sympathetic** (contraction of the pupils, narrowing of the palpebral fissure, retraction of the eyeballs, redness and increased heat of one side of the face) are at times superadded. Occasionally **paralysis of the bladder** is present, with its sequelæ and the development of **bed-sores.** With a lesion that progressively extends the clinical picture of a unilateral lesion may gradually disappear.

The symptoms of a unilateral lesion of the spinal cord may be explained by the fact that a portion of the nerve-fibers in the spinal cord are uncrossed, while others are crossed. The uncrossed fibers (motor, vasomotor, trophic, muscular and dynamic sense, and muscular sensibility) must therefore be paralyzed upon the side of the lesion, and the uncrossed (sensory fibers) upon the uninjured side.

The **prognosis** and the **treatment** will depend upon the underlying diseases.

System-Diseases of the Spinal Cord.

SINGLE SYSTEM-DISEASES.

TABES DORSALIS (POSTERIOR SPINAL SCLEROSIS; LOCOMOTOR ATAXIA).

Etiology.—Tabes dorsalis is one of the most common, and at the same time by reason of its incurable character one of the most dreaded, diseases of the spinal cord. Most cases are sequels of **syphilis.** Those syphilitics are especially liable to the disease in whom the original disorder was not thoroughly treated. Generally many years have elapsed before the symptoms of tabes appear in the sequence of syphilis, but in isolated cases I have observed the symptoms as early as the second and the third year. Not alone acquired, but at times also hereditary syphilis is believed to give rise to tabes dorsalis.

Nothing is known concerning the intimate *connection between tabes dorsalis and syphilis*. It might, for instance, be supposed that syphilis induces a lessened resistance on the part of the spinal cord to other injurious influences, which formerly were considered as the actual fundamental causes; or it is also possible that poisons (toxins) of the, as yet unknown, bacteria of syphilis cause injury to the tissues of the cord. Under either condition it must naturally be assumed that the nerve-fibers of the posterior columns of the cord or, in accordance with the most recent views, the posterior spinal nerve-roots are particularly susceptible to the injurious influences of the syphilitic poison.

Although syphilis is the principal cause for tabes dorsalis, it cannot be considered as the sole exciting factor. Other **infectious diseases** also (typhoid fever, pneumonia, diphtheria, small-pox) may act as causes. The occurrence of **toxic tabes dorsalis** also has been recorded, developing after intoxication with ergot (ergotism) or spoiled maïze (pellagra) and excessive indulgence in tobacco. At times tabes dorsalis develops in the sequence of **traumatism**, particularly concussion of the spinal column. The injurious influence of **cold** cannot be wholly denied. Upon the other hand, nothing definite is known with regard to the etiologic significance of emotional disturbances, suppression of perspiration, and the like. Tabes dorsalis occurs most frequently *between the thirtieth and fortieth years* of life, and ten times *more commonly in men* than in women.

Anatomic Alterations.—The distinctive anatomic lesion of tabes dorsalis consists in **gray degeneration of the posterior columns of the spinal cord.** This condition, however, appears to be secondary and dependent upon disease of the intervertebral ganglia, and, in addition, the remainder of the nervous system also (cerebrum, cerebellum, peripheral nerves) may be involved. The latter fact explains the variability in the clinical picture. After opening the dura mater gray bands are often visible through the translucent pia mater, traversing the entire length of the spinal cord and diminishing in size from below upward, and being obscured from view only when the pia mater upon the posterior aspect of the cord is thickened, and hence rendered opaque. The related portions of the spinal cord situated between the posterior nerve-roots and the posterior columns of the cord frequently are remarkably small and generally feel indurated (sclerotic). On *transverse section of the cord* the posterior columns appear gray and slightly translucent. While this change involves the entire extent of the posterior columns in the lumbar region, including the columns of Burdach as well as those of Goll, toward the cervical portion it gradually becomes confined to the postero-internal or columns of Goll (Fig. 80). If the changes in the spinal cord are followed from below upward, they can still be recognized in the delicate columns bounding the fourth ventricle, and at times they can be detected also in the peripheral layers of the pons Varolii and the quadrigeminal bodies. It is noteworthy that islets of medullated nerve-fibers always persist also in the most anterior portions of the posterior columns in the lumbar cord close to the gray commissure. The difference in the appearance of the anterior and the posterior *spinal nerve-roots* is striking, for while the former generally preserve their usual size and natural, medullary-white appearance, the latter are thin, gray, and almost translucent.

On *microscopic examination* disappearance of the nerve-fibers and increase of the neuroglia are found in the diseased posterior columns. In all probability the atrophy of the nerve-fibers is the primary change, to which the increase in the neuroglia is added secondarily. In the hyperplastic neuroglia, which in advanced cases acquires a fibrillated appearance, isolated fatty granule-cells and numerous amyloid bodies are not rarely present. The blood-vessels are often thickened and in places obliterated.

Anatomic alterations are encountered also in the columns of Clarke and in the zone of Lissauer, in addition to the posterior columns of the cord. In the columns of Clarke, particularly when the spinal cord is stained by Weigert's hematoxylin-method, the disappearance of the finer nerve-fibers is conspicuous, while the ganglion-cells appear unaltered. Also in the zone of Lissauer, which forms the peripheral boundary of the posterior horns. atrophy of nerve-fibers with a double contour has taken place. Disappearance of the nerve-fibers, and in lesser degree of the ganglion-cells, occurs also in the *posterior horns* of the spinal cord.

Examination of the *posterior spinal nerve-roots* discloses atrophy and disappearance of the nerve-fibers and increase of the connective tissue. Atrophic disappearance of the ganglion-cells and connective-tissue proliferation have been demonstrated likewise in the intervertebral ganglia, although the alterations in the posterior nerve-roots not rarely predominate. In the cerebral cortex and in the cerebellum disappearance of nerve-fibers, in the latter also that of the ganglion-cells and blood-vessels, has been described. Degeneration and disappearance of the ganglion-cells in the nuclei of the cerebral nerves is noteworthy, but also in individual cerebral nerves themselves degeneration and disappearance of nerve-fibers and hyperplasia of the connective-tissue occur in their trunks as well as in their intramedullary course. Degeneration and disappearance of nerve-fibers in the peripheral nerves should not be overlooked. These alterations occur particularly in the finer cutaneous branches, but are observed also in the larger nerve-trunks.

It has already been pointed out that the lesions of tabes dorsalis are dependent upon degenerative and not inflammatory alterations in the nerve-fibers of the spinal cord. It appears that this nerve-degeneration does not begin primarily in the spinal cord, but originates in the ganglion-cells of the intervertebral ganglia. As is well known, each of these ganglion-cells

gives off a process, which soon divides into two branches, one of which enters a peripheral nerve and the other the posterior column of the spinal cord. As a result of morbid activity on the part of the ganglion-cells degeneration and atrophy take place in the branch passing to the spinal cord, and these may bring about the anatomic alterations of tabes in the posterior columns. In harmony with this view is the fact that the alterations become more restricted to the columns of Goll the higher up in the cord they are situated, as fibers from the nerve-roots entering the lowermost portion of the cord become gradually displaced toward the median portion of the posterior column as they ascend. Just why the process begins in the intervertebral ganglia of the lumbar cord has not yet been determined. Only rarely does tabes dorsalis begin in the cervical cord, when the disease may be confined to the postero-external (Burdach's) columns. The condition is then designated *cervical tabes dorsalis*.

Symptoms.—Among the symptoms of tabes dorsalis a distinction should be made between constant (typical) and accidental symptoms. The former particularly are of diagnostic significance, and they alone depend upon the typical seat of the disease, while the accidental symptoms vary accordingly as in the individual case one or another portion of the nervous system becomes involved. The *typical* symptoms of tabes dorsalis include **absence of the knee-jerks, reflex pupillary immobility, swaying station,** and **ataxic gait.** *Absence of the knee-jerks,* also known as Westphal's symptom, is one of the earliest and most constant signs of tabes dorsalis, and there should always be some hesitancy in making a diagnosis of tabes dorsalis if the knee-jerks are preserved unchanged. The phenomenon is explained by the fact that in the presence of tabes dorsalis the spinal reflex arc is interrupted. This interruption was formerly believed to occur in the posterior columns of the spinal cord at the junction of the dorsal and lumbar portions, within the so-called root-entrance zone. In accordance with more recent views with regard to the development of tabes dorsalis, however, it is probable that the interruption of the spinal reflex arc occurs in the degenerated posterior nerve-roots, and the second, third, and fourth lumbar roots are those especially involved.

I have been able to demonstrate by clinical observation that also in cases of *cervical tabes* the knee-jerks may be absent if in addition to the tabes inflammation of the crural nerves also exists. In view of the great significance of absence of the knee-jerks in the diagnosis of tabes dorsalis it is exceedingly important to avoid all *sources of error* in the examination. The extremity to be examined must be perfectly relaxed, and is either crossed over the opposite member or is permitted to hang without constraint over the edge of a chair or of the bed. It is highly useful to employ the expedient of Jendrassik, the patient during the examination forcibly pulling his clasped hands apart or forcibly squeezing the finger of the examiner. By means of such devices the attention of the patient is involuntarily diverted from the examination of the knee-jerk and the member is relaxed unconsciously.

The suspicion of tabes dorsalis will be increased if in addition to absence of the knee-jerks there is also *reflex pupillary immobility.* This sign is known also, after its discoverer, as *Robertson's symp-*

tom. This is developed by having the patient look toward the sky from a bright window while light is excluded from the eyes by means of the hands held before the eyes. If now one or both hands are suddenly removed from before the eyes, contraction of the pupils on exposure to light fails to take place. The size of the pupils remains the same in the dark and on exposure to light. The sensory pupillary reaction likewise is wanting; that is, on pricking the skin dilatation of the pupil fails to take place. The reaction of the pupil in accommodation, however, is preserved, so that the pupils become dilated in distant vision, while they become contracted in near vision.

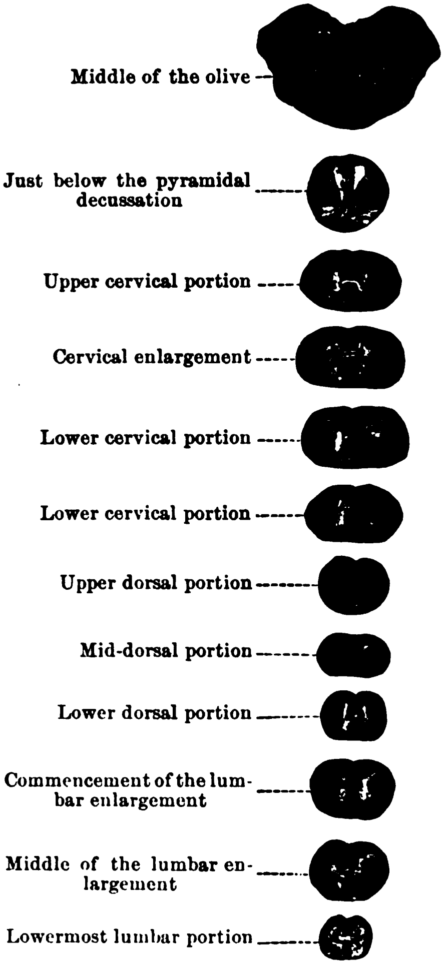


FIG. 80.—Transverse sections of the spinal cord presenting advanced degeneration of the posterior columns from a man 62 years old. The degenerated areas are light, as they appear in the spinal cord hardened in Müller's fluid.

Natural size; from transverse sections of the spinal cord hardened in Müller's fluid (personal observation, Zurich clinic).

Of less diagnostic significance is the fact that the pupils are not rarely contracted —*spinal myosis*—or are unequal.

When the eyes are closed the *body sways* markedly — ***Bracht-Romberg symptom.*** This not rarely becomes so marked that the patients are in danger of falling. Often the patient has noticed the symptom first when in washing the face he has been compelled to close his eyes.

Ataxia is so constant a symptom of tabes dorsalis that the disease has also been designated *progressive locomotor ataxia.* It can be recognized in walking from the fact that the patient holds the legs far apart and brings the feet down upon the heels, raising the extremities unnecessarily high, and at the same time making awkward and jerky movements. The gait is also appropriately characterized as *stringhalt-like.* If the patient be requested to place one foot in advance of the other in walking, or if he make an attempt to walk upon a crack in the floor or upon a chalk-line, he will be able to do any of these with extreme difficulty and awkwardness, if at all. On lying in bed the ataxia of the legs will be shown by the fact that in elevating one of the members it will oscillate to and fro and be thrust beyond the middle line toward the opposite side. All ataxic disturbances are increased in the dark, when the patient is unable to guide the movements with the aid of vision. Ataxic movements are not uncommon also in the arms. They may be readily recognized if the arms are widely separated when the eyes are closed and an attempt is made to bring the finger-tips together. In ataxic patients to-and-fro movements in the air will follow for some time before the finger-tips are brought in contact.

Much discussion has taken place as to the *mode of origin of the ataxia.* In our opinion the view of Leyden is the most likely, and according to which the ataxia is the result of sensory disturbances, not alone cutaneous sensibility, but particularly also the sensibility of the deeper structures (muscles, tendons, fascia, joints), being affected.

Possibly disturbances in the reflex paths in the spinal cord may be operative. Some clinicians have attributed the ataxia to disease of definite spinal tracts supposed to possess coördinative activity (lateral cerebellar tracts), but nothing is known with regard to the existence of such tracts.

The typical symptoms of tabes dorsalis thus far considered develop slowly, and generally with **prodromes.** The latter include with especial frequency pain in the course of one or both sciatic nerves, at times attacks of headache and vomiting, vertigo, and the like.

Among the *accidental symptoms* of tabes dorsalis sensory disturbances particularly predominate, and this can be readily understood from the fact that the morbid process develops especially in the sensory sections of the cord; but motor, trophic, secretory, and vasomotor manifestations, together with visceral disturbances, also occur not at all rarely. In view of the great variability in the symptoms, a brief consideration of the most important of them must suffice. **Sensory disturbances** are scarcely ever wholly absent. Often the patients complain of **paresthesiæ**, particularly the crawling of ants, prickling, burning, a sense of cold, and shooting pains. Often complaint is made of a painful girdle-sense about the trunk, and but seldom of pain in the vertebral column itself. Cutaneous **anesthesia** is frequently encountered, at times total, at other times partial. The anesthesia is generally most pronounced on the soles of the feet, and progressively diminishes toward the trunk. Often the patients complain of not feeling the floor beneath their feet, and in walking they appreciate a [illegible] upon wool, or were going about [illegible]

[illegible] application of a needle upon the [illegible] often the *conduction* of sensation [illegible] seconds elapsing before the [illegible] of a needle. *Duplicate sensations* [illegible] a single needle-prick is felt by the patient [illegible] pain or at first slight and after [illegible] of pain occurs. Not rarely the [illegible]

[illegible] of the **muscular sense** are demonstrable, [illegible] the patient is incapable of determining [illegible] members placed at different levels is the [illegible] and the like. **Paralysis** plays [illegible] clinical picture of tabes dorsalis. [illegible] occurrence of the ataxia, [illegible] power in the lower extremities [illegible]. At times paralysis in the distribution [illegible] appears early. This has been [illegible] which may gradually subside and [illegible].

[illegible] twitching are less common manifesta[illegible]

[illegible] **trophic disorders** the *tabic articular changes* especially [illegible] attention. The large joints are attacked with [illegible] frequency, particularly the knee-joints. As a rule, the [illegible] enlarged, and they are at the same time but little, if at all, tender to touch, and after the articular enlargement has subsided deformity and ankylosis may persist because the articular [illegible] are greatly distorted.

Among other trophic disorders in the course of tabes dorsalis there may be mentioned *abnormal fragility of the bones*, an *undue tendency to laceration on the part of certain tendons*, particularly the Achilles tendon, *hemiatrophy of the tongue, perforating ulcer of the foot*, *herpes zoster*, *thickening af the nails, loss of the teeth, the nails*, and *the hair*, etc.

Vasomotor and *secretory disorders* are revealed by unusual redness and heat of the skin, by hyperidrosis, increased secretion of saliva and of tears, and the like. Among the *visceral disturbances* of tabes dorsalis the **visceral crises** deserve first mention. These consist in sudden attacks of pain and functional disturbance in certain organs. *Gastric crises* are the most common and the best known. These consist in attacks of severe epigastric pain with profuse vomiting, which may persist for days, and is at times attended with an excess of hydrochloric acid. If the attacks are repeated in quick succession, the patient may become exhausted in an alarming degree. Often such patients are treated for years for chronic gastric catarrh, and are sent each summer to health-resorts before the real nature of the gastric disorder is recognized. At times gastric crises deviate from the typical picture described in so far as they are attended either with severe epigastric pain alone or only with profuse vomiting without such pain.

Among other visceral crises *intestinal crises* may be mentioned. These are attended with paroxysmal attacks of diarrhea, usually accompanied by colic. *Rectal crises* are attended with burning pain in the rectum. *Renal crises* suggest the clinical picture of renal colic, and are attended with severe pain in the loin, but they are generally unattended with albuminuria and hematuria. *Laryngeal crises* are characterized by attacks of cough and dyspnea resembling whooping-cough or laryngismus stridulus. *Pharyngeal, bronchial, cardiac, clitoral,* and *urethral crises* also have been described.

Among other visceral manifestations **ocular** changes deserve first place on account of their diagnostic importance. Not rarely *optic atrophy* develops, and may give rise to gradual blindness. At times blindness occurs early, so that the patients must bear the burden of their disease for many years in a blind state. *Paralysis of the ocular muscles* is often present, and it may disappear after a time, being probably more frequently dependent upon peripheral neuritis than upon disease of the nuclei of the nerves. **Paralysis of the laryngeal muscles** also is not rarely observed in the course of tabes dorsalis. The statement that tabic patients present **valvular lesions of the heart** with especial frequency I am unable to confirm from personal observation. Frequently, and at times early, symptoms of **paralysis of the bladder** occur. These may appear and disappear repeatedly in the course of the disease. Eventually they may persist, and then are attended with the dangers of urinary infection and of bed-sores. In men **impotence** not rarely occurs early. Women, however, may, in spite of advanced tabes dorsalis, conceive and bear children in the usual manner.

The *course of tabes dorsalis* is chronic, and the disease often extends over many years. Attempts have been made to distinguish several stages of the disease, although all such divisions are somewhat artificial and of slight practical value. Should the ataxia in the lower extremities become excessive, the patients will be unable to move about alone, even with the aid of a cane, and will be compelled to remain constantly in bed. At times in attempts at walking the patient may stumble over his own feet and fall to the ground. After the patient has been confined to bed for a long time atrophy of the muscles of the lower extremities develops from disuse, with a condition of weakness and the development of *muscular contractures*. At times *apoplectiform attacks* occur with hemiplegia, the latter disappearing; but if dependent upon thrombosis of cerebral arteries the paralysis may persist. The *mental state* of the patient is often stated to be an abnormally cheerful one. I should rather describe it as one of fatalistic resignation. *Psychic disturbances* occur, and predominate from the outset in cases in which tabes dorsalis occurs in connection with progressive paralysis of the insane. Death takes place in consequence either of accidental disease (pneumonia, pulmonary tuberculosis) or of progressive exhaustion, or of urinary infection or septicemia; at times also with the occurrence of a soporose state of sudden onset.

Diagnosis.—In the diagnosis of tabes dorsalis reliance should be placed upon the four typical symptoms: Absence of the knee-jerks, reflex pupillary immobility, unsteadiness in station, and ataxia. Confusion with **peripheral pseudotabes** may readily occur when this condition develops as a result of polyneuritis. Peripheral pseudotabes occurs with particular frequency and distinctness in connection with alcoholic, diabetic, postdiphtheric, and toxic polyneuritis. Although the reaction of the pupils may be exceedingly sluggish in the presence of polyneuritis, reflex pupillary immobility scarcely ever occurs, and the presence of the latter, therefore, is always indicative of tabes dorsalis. Tabes dorsalis will scarcely be confounded with **cerebellar ataxia**, because the knee-jerks are preserved in the presence of cerebellar disease. **Hereditary ataxia** often develops during childhood, and is attended with nystagmus and articulatory disturbances.

Prognosis.—The prognosis of tabes dorsalis is unfavorable with regard to recovery, although life does not appear to be immediately threatened, and if the patient does not come under professional observation too late material improvement may at times be brought about.

Treatment.—As syphilis is the cause of tabes dorsalis in the majority of cases, it might be supposed that in such cases **causal therapy** with mercurial inunctions (5.0—75 grains—daily) and potassium iodid (5.0 : 200—75 grains : 6½ fluidounces; 15 c.c.—1 tablespoonful—thrice daily) would yield good results. Too much, however, should not be expected in this connection. Improvement fails to take place in the majority of cases, or under the most favorable circumstances it is but slight. This is probably due to the fact that the patients generally come under professional observation too late, and that under the most favorable circumstances preparations of mercury and of iodin are capable only of arresting the morbid process or checking the course of the disease, and not of effecting recovery. Such nerve-fibers as are already degenerated are lost forever. Nevertheless, antisyphilitic treatment should always be advised, and it should even be repeated from time to time. Among **internal remedies**, I have in a number of cases observed surprising results from the use of *silver nitrate*, while the remaining large number of nervines have been disappointing:

℞ Silver nitrate, 0.3 (4½ grains);
Aluminum hydrate, sufficient to make 30 pills.
Dose: 1 pill thrice daily

Gymnastic exercises with the lower extremities, and if necessary also with the upper extremities, are to be warmly recommended. The patient is, for instance, instructed to walk several times daily upon certain lines and figures, and as a result the ataxic disturbances not rarely subside, and the power of locomotion is greatly improved. I have in a number of cases also observed material improvement in the power of locomotion as a result of treatment by **suspension.** Not much can be accomplished by courses of treatment at the **baths** (saline, sulphurous, indifferent baths, courses of treatment with cold water). Well-to-do patients may be advised to pass the winter in mild climatic resorts (Riviera), in order to be in the open air as much as possible. A **sea-voyage** also may be recommended. For neuralgias, antipyrin (1.0—15 grains—thrice daily) or phenacetin (1.0—15 grains—thrice daily) may be employed. At times **subcutaneous injections of morphin** must be resorted to. These constitute also the most reliable means of relieving severe visceral crises. The *faradic* and the *galvanic current* will not afford any material relief.

SPASTIC SPINAL PARALYSIS.

Etiology and Anatomic Alterations.—*Disease of the lateral pyramidal tracts* is believed to be the cause of spastic spinal paralysis. Although the clinical picture of spastic spinal paralysis is by no means rare, it has been shown that it generally results in consequence of other **diseases of the spinal cord,** while it occurs with exceeding rarity as an independent disorder. It is encountered with especial frequency in connection with myelitis, spinal pressure-paralysis, multiple cerebrospinal sclerosis, combined system-disease of the spinal cord, and syphilitic spinal paralysis. Spastic spinal paralysis develops not rarely in children when in consequence of protracted or artificial labor, or during teething, or after infectious diseases cerebral paralysis occurs, at times in the form of monoplegia, at other times of hemiplegia, and at still other times of paraplegia. The condition is known also as spastic rigidity of the extremities or Little's disease. **Exposure to cold, infectious diseases,** and **alcoholism** are mentioned as causes of independent spastic spinal paralysis, although actually little of a definite nature is known in this connection.

Symptoms, Diagnosis, and Prognosis.—The symptoms begin generally in the lower extremities. Muscular weakness develops, and may progress to marked **paralysis.** At the same time **muscular contractures** appear, particularly in the extensors and the adductors of the thigh and in the flexors of the foot. The gait assumes the character that has been designated **spastic-paretic.** The **tendon-reflexes** are abnormally increased. Sensibility and the functions of the bladder and the rectum, however, remain undisturbed.

The *spastic-paretic gait* is characterized by a peculiar rigidity of the lower extremities, which in walking are applied firmly to the ground like stilts. The extremities are scarcely flexed at the hip and the knee, and the thighs can be moved in front of each other only with great difficulty on account of adductor-contracture, so that often the pelvis is greatly rotated in walking. Contractures of the muscles of the calf often give rise to pes equinus, so that the patient in walking first applies the toes to the ground. and in consequence of exaggeration of the tendon-reflexes the legs are set into tremulous and agitated movement. If the morbid process advances, contractures gradually develop in the flexors of the thigh and the leg, and these render walking wholly impossible.

Exaggeration of the knee-jerks will be manifested by marked and persistent contraction in the extensor of the thigh after even a slight blow with the plexor. At times reflex muscular twitching occurs as well in members not percussed. *Ankle-clonus* also is exaggerated ; and if with the leg extended dorsal flexion of the foot is practised suddenly, active flexion and extension of the foot take place, which not rarely can be prevented intentionally by rapid dorsal flexion of the great toe.[1] If the arms are involved, active reflex muscular contraction will follow percussion of the extensor tendons of the forearm or the tendon of the triceps. The *periosteal reflexes* also are abnormally increased.

The *duration* and the *curability* of the symptoms depend upon the nature of the primary disorder. In cases of independent spinal paralysis complete recovery has been observed at times in the course of a few weeks.

Treatment.—Treatment should be directed, in the first place, to the primary disorder—**causal therapy**. In cases of independent spastic spinal paralysis hot baths, nervines, electricity, and massage have been employed.

DISEASE OF THE GANGLION-CELLS OF THE ANTERIOR HORNS (ANTERIOR POLIOMYELITIS).

Poliomyelitis indicates only inflammation in the anterior horns of the spinal cord, but the structures upon which the characteristic features of the clinical picture depend are the multipolar, motor-trophic ganglion-cells in the anterior horns, whose destruction is followed by *flaccid paralysis* of the related muscles, rapidly developing *degenerative muscular atrophy*, *degenerative electric reaction* in the diseased nerves and muscles, *abolition of the reflexes*, with *preservation of cutaneous sensibility and of the functions of the bladder and the rectum.* In accordance with the course of the disease and the age of the patient, several clinical varieties may be distinguished, namely: acute spinal paralysis of childhood; acute, subacute, and chronic spinal paralysis of adults; spinal progressive muscular atrophy.

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[1] Irritation of the sole of the foot induces extension of the great toe and perhaps also of the remaining toes.—A. A. E.

ACUTE SPINAL PARALYSIS OF CHILDHOOD.

Etiology.—Acute spinal paralysis of childhood is one of the *most common diseases* of this period of life, and experience has shown that it is more common in *boys* than in girls, and that it occurs especially at the period of *dentition*, and from that time on to the fourth year of life. There is a tendency at the present day, and not without reason, to assume the occurrence of infection in many cases, as in the first place the entire course of the disorder corresponds in many respects with that of an acute infectious disease, and in addition the disease occurs at times in endemic or epidemic distribution. Besides, **exposure to cold, traumatism, fright,** and **emotional disturbances** generally, **difficult dentition,** and **antecedent infectious disease** are assumed to be causative factors.

Anatomic Alterations.—The alterations of acute spinal paralysis of childhood arise from the blood-vessels of the anterior horns. These are dilated and extremely tortuous, ruptured in places, and presenting in their adventitial lymph-sheaths, as well as outside of these, accumulations of round cells. The ganglion-cells are swollen, plump, in part deprived of their processes, and they are likewise surrounded by round cells. In the fresh state the inflammatory area presents a blood-red and softened appearance. Subsequently it becomes converted into a sort of cicatricial, sclerotic, and contracted tissue, which contains but a few, if any, ganglion-cells, and may persist throughout the rest of life. Even after the lapse of many years the disease-focus is recognizable from the contraction and diminution in size of the anterior horn. The disease is at times unilateral and at other times bilateral, and it may appear as a small, circumscribed focus or as an extensive process, or as a number of disseminated foci. In the **anterior nerve-roots** related to the foci of disease degeneration and disappearance of nerve-fibers are found—alterations that may be traced in the peripheral nerves to the muscles. In the **muscles** atrophy of muscular fibers takes place, with increase in the connective tissue, the latter being not rarely well supplied with fat-cells. The atrophic muscular fibers are characterized by an abundance of sarcolemma-nuclei, and often also by unusual distinctness of their transverse striation. Other muscle-fibers exhibit fibrillation and vitreous degeneration. In addition to atrophic muscle-fibers, here and there also hypertrophic fibers are present, which not rarely contain vacuoles and accumulations of nuclei.

e paralyzed member presents a cyanotic appear- lly feels cool. **Trophic disturbances** likewise have

The skin of the paralyzed parts frequently pre- ally thick layer of adipose tissue, which in part arked wasting of the paralyzed muscles. Also, in emselves active deposition of fat takes place, which izes the loss of actual muscular tissue. It is note- hat the bones are notably retarded in growth, are ually brittle, and when the child has grown up the mber at times forms only a small appendage. Even se, and blood-vessels in the distribution of the mus- ls fail to grow normally.

ional activity of the *bladder* and the *rectum* does not

ular contractures develop frequently in the course of nd they may give rise to marked deformities and in- th the function of the paralyzed members. Abnormal n the healthy muscles, the weight of the bed-cloth- ilar circumstances are the causative factors for such ortening. Under such conditions club-foot of any develop, as well as spinal curvature, flail-joint, and oor patients may pass through life as cripples, and infirmity as a means for exciting the sympathy of ter circumstances. They often devise ingenious appa- e purpose of neutralizing the functional disturbance in the arms, as, for instance, supports, upon which they out, rolling vehicles, and the like.

se of the disease is chronic, and the patients often attain ge. Occasionally symptoms of *spinal progressive mus- hy* develop at a later period. Other patients become epileptic. Some are greatly burdened by excessive which many patients are predisposed by sedentary pur- eficient activity.

osis.—The recognition of acute spinal paralysis of s easy. The disease might most readily be confounded **polyneuritis**, but with the latter nerves and muscles are ensitive to pressure, and, in addition, various sensory s are present. **Parturitional** or **obstetric palsies** (p. 499) ed from birth, and in cases of **spastic spinal paralysis** -reflexes are exaggerated.

osis.—Although, as a rule, the disorder is unattended er to life, nevertheless the prognostic outlook is not because the paralysis does not subside and muscular s and deficient growth of the bones increase the in-applied to the head, and, if the child can swallow, **phenacetin** (0.3—4½ grains—twice daily) or **antipyrin** (0.3—4½ grains—twice daily) administered internally. The application of an **ice-bag to the vertebral column** may also be advised. For the paralysis, **massage** should be practised daily. At the same time varied passive movements of the joints should not be omitted, in order to prevent as much as possible the development of contractures. The application of **electricity** to the spinal cord and to the paralyzed muscles will be attended with as little success as the internal administration of the various **nervines.** Little also is to be expected from **baths** (saline or sulphurous or indifferent thermal baths). On the other hand, **surgical** and **orthopedic measures** may yield good results in the presence of contractures.

ACUTE, SUBACUTE, AND CHRONIC INFLAMMATION OF THE GANGLION-CELLS OF THE ANTERIOR HORNS IN ADULTS (ACUTE, SUBACUTE, AND CHRONIC POLIOMYELITIS OF ADULTS).

Acute anterior poliomyelitis of adults in its clinical course most closely resembles acute spinal paralysis of childhood. It is, on the whole, a *rare disease*, which experience has shown is *more common in men* than in women, and which generally occurs *before the thirtieth year of life.* It has been observed in the sequence of *infectious diseases* (puerperal fever, gonorrhea). I have had under observation individuals in whom the affection gave the impression of being an *independent infectious disease. Exposure to cold, traumatism, over-exertion,* and *alcoholic* and *venereal excesses* also are capable of acting as causes. The disease begins with *febrile prodromes.* Often a chill occurs at the beginning, followed by high fever, stupor, and delirium—a condition that often persists for one or two weeks. Clonic convulsions are, however, commonly absent, perhaps because the motor cortical centers are more resistant in adults than in children. *Muscular paralysis* then generally sets in suddenly, and, as in cases of spinal paralysis of children, it is of flaccid type, is attended with rapid degenerative wasting, degenerative electric reaction, and increased mechanical irritability, with loss of the cutaneous and tendinous reflexes, while sensibility and the functions of the bladder and the rectum exhibit no derangement. At times the paralyzed nerves and muscles are sensitive to pressure, and the overlying skin is often mottled blue and feels cold. Further, the paralysis at times subsides in part—*temporary paralysis*—and in individual rare cases the paralysis has disappeared entirely. The paralysis disappears especially from such muscles as exhibit no degenerative electric reaction. The *distribution of the paralysis* varies, as with spinal paralysis of childhood, between monoplegia, paraplegia, hemiplegia, and paralysis of individual neuromuscular areas. Cerebral nerves are but rarely affected. After the paralysis has existed for some time *muscular contractures* may develop. Disturbances in growth of bones, tendons, fasciæ, and blood-vessels do not occur, because the members of the patient are fully developed.

The **diagnosis,** the **prognosis,** and the **treatment** are the same as for acute spinal paralysis of childhood.

Subacute and chronic anterior poliomyelitis of adults is due to the same *causes* as the acute variety, is likewise one of the less common diseases of the spinal cord, and develops generally *between the thirtieth and the fiftieth year of life.* The disorder sets in insidiously without antecedent febrile manifestation, most commonly in the extensors of the leg, then slowly invading the muscles of the trunk and the arms, and even those supplied by the cerebral nerves. Less commonly the onset is marked by paralysis in the arms, and this may then extend to the legs. The disease is progressive in course, although it may happen that the paralysis subsides spontaneously in part or at times even entirely—temporary paralysis; although, on the other hand, danger exists that the disease may involve cerebral nerves (hypoglossal, vago-accessory, glossopharyngeal), and death result from inspiration-pneumonia or paralysis of respiration or of cardiac action. Accordingly as the muscular paralysis develops within a few weeks or many months a distinction is made between subacute and chronic poliomyelitis. The character of the paralysis is that already described for anterior poliomyelitis (flaccid paralysis, rapid degenerative muscular atrophy, degenerative electric reaction, abolished reflexes, preservation of cutaneous sensibility, retained functional activity of the bladder and the rectum). When the paralysis has existed for some time *muscular contracture* may develop. The *duration of the disease* may extend over four years.

The **anatomic alterations** consist in disappearance of the large motor-trophic ganglion-cells in the anterior horns. In addition, changes in the blood-vessels (thickening of the wall, nuclear multiplication and hyperplasia of the neuroglia) may occur. Degeneration and atrophy of nerve-fibers also have been found in the anterior nerve-roots and in the peripheral nerves.

In some cases areas of degeneration have been found in some columns of the cord and disappearance of ganglion-cells in the columns of Clarke.

The **diagnosis** is not always easy. Subacute and chronic anterior poliomyelitis is distinguished from *spinal progressive muscular atrophy* by the fact that progressive muscular atrophy generally begins in the muscles of the hand, that atrophy of the muscles precedes paralysis, and that the atrophy involves the muscles only in bundles (fascicular), and not totally as in cases of anterior poliomyelitis. *Amyotrophic lateral sclerosis* is attended, in addition to muscular wasting and contractures, especially with exaggeration of the tendinous and periosteal reflexes. Finally, in the presence of progressive *polyneuritis* sensory disturbances are generally present in addition to motor disorders.

The **prognosis** and the **treatment** are the same as for acute spinal paralysis of childhood.

SPINAL PROGRESSIVE MUSCULAR ATROPHY.

Etiology.—Little of a definite nature is known with regard to the etiology of spinal progressive muscular atrophy. The disease, which is uncommon, occurs more frequently in *men*, and, as a rule, develops only *after the thirtieth year of life*. Among the causative factors **exposure to cold, over-exertion, traumatism,** and **antecedent infectious disease** have been mentioned. In exceptional instances spinal progressive muscular atrophy has been observed in persons that in early life had suffered from **spinal paralysis of childhood.** On the other hand, *heredity*, in contradistinction from the myopathic form of progressive muscular atrophy, is not of especial etiologic significance.

Anatomic Alterations.—There is a great deficiency in good and reliable anatomic descriptions. The disease is characterized by disappearance of the **large motor-trophic ganglion-cells** in the anterior horns of the spinal cord. At the same time the ganglion-cells frequently become filled with yellowish or yellowish-brown, granular pigment, undergo progressive contraction to small roundish structures without processes (so-called pigmentary degeneration of the ganglion-cells), and finally undergo complete destruction, while neuroglia takes their place. The destruction of each ganglion-cell is necessarily followed by degeneration of the entire spinal-peripheral neuron, and accordingly degeneration and atrophy of the nerve-fibers can be traced in the related *anterior nerve-roots* of the spinal cord and in the *peripheral nerves* to the muscles. The *diseased muscles* exhibit marked atrophy and a light, usually brownish color. The loss of individual muscle-fibers can be seen with especial distinctness on transverse section, while the interstitial connective tissue is abnormally increased and generally contains many nuclei. In many places muscle-fibers are replaced by remains of granular, yellow or brownish pigment. Here and there abnormally dilated blood-vessels with thickened walls can be seen. In addition to atrophic muscle-fibers a greater or lesser number of hypertrophied muscle-fibers are present, and which not rarely exhibit vacuoles and nests of nuclei.

The disappearance of the muscular fibers occurs in various ways. Muscular fibers can be seen that are characterized by marked diminutiveness, distinct transverse striation, and an abundance of sarcolemma-nuclei. Other muscle-fibers, on the other hand, exhibit granular turbidity, fatty degeneration, fibrillary or discoid disintegration or fibrillation as in the case of vitreous muscular degeneration.

Symptoms.—Muscular atrophy and the muscular weakness and muscular paralysis dependent thereon generally appear first in the small muscles of the hand, in the thenar and hypothenar eminences, in the interosseous and the lumbrical muscles. Then the extensors upon the dorsal aspect of the forearm are involved, next the deltoid muscle, and only subsequently the muscles of the upper arm. The process may gradually extend to the muscles of the trunk. The muscles of the leg either remain exempt or are involved only late. Not rarely the disease extends from the ganglion-cells of the anterior horns of the cervical portion of the spinal cord to the ganglion-cells of the nuclei of the cerebral nerves on the floor of the fourth ventricle, which have the same functions as the ganglion-cells of the spinal cord. The nuclei of the hypoglossal, vago-accessory, glossopharyngeal, and facial nerves especially are involved, and, accordingly, to the symptoms of spinal progressive muscular atrophy the clinical picture of chronic progressive bulbar paralysis is superadded. Naturally, the reverse may happen; that is, progressive bulbar paralysis develops first, the disease of the ganglion-cells extending from the nuclei of the cerebral nerves to the ganglion-cells of the cervical cord, with the development secondarily of spinal progressive muscular atrophy.

The muscles of the right hand are involved somewhat earlier than those of the left. Only in left-handed persons and in those whose work requires greater use of the left hand does the disorder begin in this hand. In either event the other hand also is soon involved, and the disease then exhibits a symmetric distribution. The disorder usually sets in insidiously. The patients generally note at first undue readiness of fatigue and awkwardness in the movements of the fingers, and on more careful examination it will be found that the thenar and hypothenar eminences have lost their fulness and are flattened, so that the subjacent metacarpal bones can be readily reached. The thumb is often abducted and extended, so that the so-called ape's hand results, as long as the extensors of the thumb and the long adductor upon the dorsal aspect of the forearm remain intact. Interosseous spaces of unusual depth upon the dorsum of the hand result from atrophy of the interosseous muscles, and disappearance of the lumbrical muscles will be disclosed by apparent excavation of the palm of the hand and prominence of the tendons of the flexors of the fingers beneath the skin of the palm. Weakness of the interossei often gives rise to the **claw-hand.** The more marked the muscular atrophy the greater is the weakness of the wasted muscles and the more pronounced is the functional disturbance in the fingers.

Muscular wasting in the extensors upon the dorsal aspect of the forearm gives rise to an appearance of emaciation and flattening. The interosseous space between the ulna and the radius often is distinctly recognizable as a deep depression, and the borders of the radius and the ulna can be readily palpated (Fig. 81). The supinators long remain intact, undergoing atrophy only when the biceps and the internal brachial in the upper arm are involved. In the presence of atrophy of the deltoid the region of the shoulder appears flattened, and the head and the neck of the humerus are readily accessible through the skin. At times a depression between the head of the humerus and the acromion process is appreciable on ocular inspection. This is indicative of a flail-joint, which results from the fact that the deltoid is so greatly weakened in its powers that the forearm by reason of its weight drops downward and its

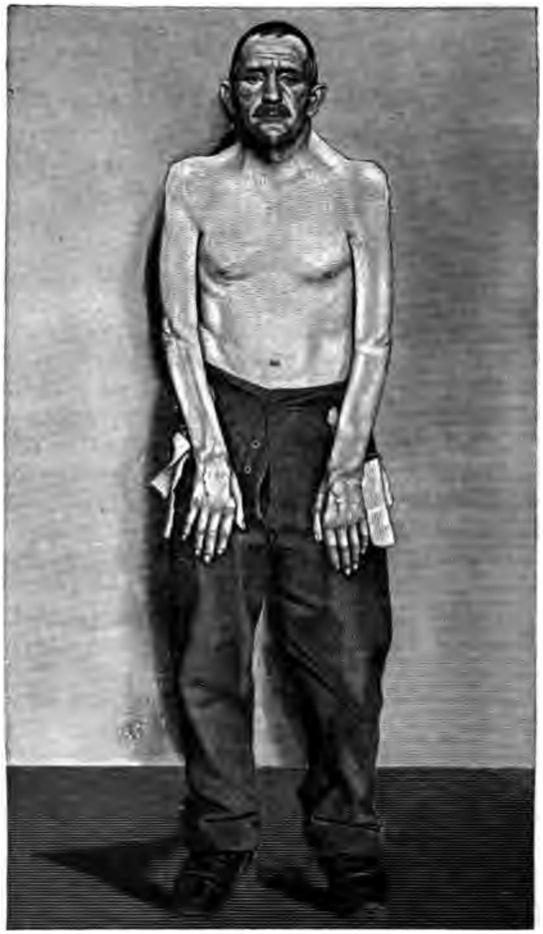

FIG. 81.—A man, 52 years old, with spinal progressive muscular atrophy; from a photograph (personal observation, Zurich clinic).

articular head is separated from the glenoid cavity. The weakness of the deltoid muscle is characterized particularly by an inability to raise the arm to the horizontal level at all perfectly or for more than a short time. Of the muscles of the upper arm, the triceps remains uninvolved the longest.

An exceedingly common symptom of spinal progressive atrophy is **fascicular (fibrillary) muscular contraction.** This consists in suddenly occurring and rapidly disappearing muscular twitching, involving isolated muscle-bundles. At times it is so persistent and distributed among so many muscles that these appear to be in a condition of constant restlessness. In other cases, however, a long time elapses before the muscular twitching becomes visible. It can often be induced artificially by percussion, by blowing upon the skin or spraying it with cold water. Nothing of a definite nature is known with regard to its development. It occurs transiently also in healthy persons under the influence of cold, as, for instance, on removal of the clothing. **Degenerative electric reaction** is present in the atrophied muscles, although the examination requires skill and attention, as the atrophy, and accordingly also the degenerative electric reaction, are often present only in bundles of muscles. All **reflex movements** (cutaneous, tendinous, and periosteal reflexes) are wanting, or at least are enfeebled, in the distribution of the affected muscles. **Cutaneous sensibility** remains unchanged, but not rarely the diseased nerves and muscles are sensitive to pressure. **Vasomotor disturbances** occur frequently, and give rise to cyanotic discoloration and coldness of the skin, particularly in the fingers, hands, and forearms. At times **trophic disturbances** occur, as, for instance, thickening and brittleness of the skin, abnormal growth of hair, pemphigus, herpes, thickening of tendons or joints. The functions of the **bladder** and the **rectum** remain undisturbed.

The disease pursues an extremely slow *course* and sometimes extends over many years. The greater the number of arm-muscles that waste and become incapacitated functionally the greater is the degree of helplessness of the patient. *Muscular contractures* also develop gradually. If the extensors of the back are involved in atrophy and paralysis, the patient eventually will be unable to assume the erect position unaided. If *bulbar symptoms* are superadded, the tongue undergoes wasting, becomes movable with difficulty, and exhibits fibrillary contractions, and difficulties in speech and in the formation of a bolus develop as a result of hypoglossal paralysis. Difficulty in swallowing, paralysis of the laryngeal muscles, and acceleration of the action of the heart occur (paralysis of the glossopharyngeal and vago-accessory). The muscles of the face, and especially those of the lips and the cheeks, become thin, atrophied, and stiff, and movable with difficulty (facial palsy). The complication is of serious significance, because death may result rapidly in consequence of inspiration-pneumonia or paralysis of respiration or of cardiac action.

Diagnosis.—The diagnosis of spinal progressive muscular paralysis can scarcely be considered difficult. Confusion might most likely occur with **myopathic** and **neural progressive muscular atrophy**; that is, with those varieties of progressive muscular wasting and paralysis dependent upon disease of the muscular structure itself or of the peripheral nerves. In the differential diagnosis it should be borne in mind that myopathic and neural progressive muscular atrophy are frequently *hereditary*, begin at times in *early life*, and frequently commence in the muscles of the legs or the facial muscles, or in the muscles of the shoulder-girdle. The same muscular changes as occur in spinal muscular atrophy are observed also in *syringomyelia*, but the latter is attended besides with partial anesthesia and trophic disorders. Spinal progressive muscular atrophy is distinguished from **subacute** and **chronic anterior poliomyelitis** by the fact that in the former muscular atrophy appears first, and then weakness and paralysis of the atrophic muscles while in poliomyelitis paralysis appears first, and then atrophy of the paralyzed muscles occurs. *Chronic polyneuritis* does not pursue the typical course of spinal progressive muscular atrophy, and it is generally attended also with sensory disturbances. Occasionally muscular atrophy complicates antecedent **arthritis.** Under such circumstances only a few muscles in the vicinity of the inflamed joint are involved.

Prognosis.—Spinal progressive muscular atrophy is an incurable disease of progressive course and distribution. The prognosis is therefore unfavorable. Life naturally may be preserved for many years, particularly if bulbar symptoms do not occur.

Treatment.—Internal remedies are without effect. In the first place, **massage** of the affected muscles may be recommended. At least, no more can be accomplished by **electric treatment** of the muscles. **Baths** (indifferent, saline, sulphurous) have also been employed.

COMBINED SYSTEM-DISEASES OF THE SPINAL CORD.

HEREDITARY ATAXIA.

Etiology.—Hereditary ataxia is an uncommon disease, first carefully studied by Friedreich, and therefore known also as *Friedreich's ataxia*. The disorder is generally an *hereditary* or *familial* one, and boys are more commonly attacked than girls. **Mental disease** and **alcoholism** in the parents, as well as **antecedent infectious disease**, have been named as causative factors.

Symptoms and Diagnosis.—The disease generally begins in childhood (between the third and the seventh year) or at the period of puberty. The most conspicuous symptom is the markedly *ataxic gait*, which is suggestive of the staggering of an intoxicated person. Marked swaying of the body is noticeable particularly on standing—**static ataxia.** The ataxia soon extends from the legs to the arms. In walking the head is involved in active oscillation. The muscles of articulation and the ocular muscles also are involved in the ataxia, with the development of **ataxic anarthria** and **ataxic nystagmus. Swaying of the body** when the eyes are closed is frequently, though not invariably, present. The **knee-jerk** is almost always absent. The *foot* often exhibits deformities, and the great toe is generally in a position of dorsal flexion. **Spinal curvature** also is frequent. In contradistinction from ordinary tabes dorsalis sensory disturbance, visceral crises, paralysis of the bladder and the rectum, trophic changes, reflex immobility of the pupils, paralysis of ocular muscles, and optic atrophy are almost always wanting. The disease pursues a *chronic course*, and at times may extend over more than thirty years. Death usually results from some accidentally intercurrent disease.

Anatomic Alterations.—Disappearance of nerve-fibers and proliferation of neuroglia are found in the spinal cord in the posterior columns, in the lateral pyramidal tracts, in the lateral cerebellar tracts, and in the tracts of Gowers (Fig. 82). Disappearance of ganglion-cells and of nerve-fibers has been observed also in the columns of Clarke. The posterior spinal nerve-roots are often thin, atrophic, and gray. Nerve-degeneration has been observed also in peripheral nerves. Recently attention has been called by several observers to atrophy of the cerebellum, and there is a tendency to ascribe the staggering gait to this condition (cerebellar ataxia). There is much in favor of the view that the anatomic alterations result from defective development of individual portions of the spinal cord and the cerebellum.

Prognosis and Treatment.—There can be no hope of therapeutic success, and the prognosis is therefore unfavorable with regard to recovery.

AMYOTROPHIC LATERAL SCLEROSIS.

Etiology.—Amyotrophic lateral sclerosis is a *rare disease*, which is observed *more commonly in women* than in men, and generally appears *between the thirty-fifth and the fiftieth year of life*. **Exposure to cold, traumatism,** and **emotional disturbances** have been mentioned as causative factors. Lead-poisoning also is thought to be responsible for the disease. Isolated observations have been made pointing to the *hereditary* and *familial occurrence* of the disorder.

Anatomic Alterations.—The affection represents a disease of the **motor-lateral pyramidal tracts and the motor-trophic ganglion-cells in the anterior horns of the spinal cord,** and accordingly consists in a combination of the symptoms of lateral sclerosis and those of chronic anterior poliomyelitis. The disease of the lateral pyramidal tracts, which consists in loss of nerve-fibers and increase of neuroglia, occurs first, and may at times be followed toward the brain throughout the entire motor or corticomuscular path to the motor ganglion-cells of the cerebral cortex. Likewise the disease of the ganglion-cells of the anterior horns (atrophy, disappearance of ganglion-cells, and proliferation of neuroglia) causes degeneration and atrophy of nerve-fibers in the anterior spinal nerve-roots, and in the peripheral nerves to the muscles. The muscles themselves also may exhibit degenerative atrophy. The entire motor path from the cerebral cortex to the peripheral muscle termination is thus involved in cases of amyotrophic lateral sclerosis, and the alterations consist in degenerative atrophy of the corticospinal and spinal-peripheral motor neurons. In isolated cases the anterior direct pyramidal tracts and even the [illegible] also have been found degenerated. Disappearance of [illegible] of the posterior horns of the spinal cord has [illegible] been observed.

Symptoms and Prognosis.—In accordance with the motor [illegible] manifestations consist of a combination of [illegible] spastic spinal paralysis [illegible] [illegible] forearm, invades the deltoid and other muscles of the upper arm, and latest, if at all, involves the flexor-muscles of the forearm. It is noteworthy that the muscles do not undergo fascicular atrophy or wasting in bundles, but throughout their entire extent. The atrophic muscles exhibit **fascicular (fibrillary) contractions** and **degenerative electric reaction.**

Should the changes in the spinal cord extend downward from the cervical portion, the patients complain of **stiffness in the back** if the muscles in this situation become involved. Involvement of the muscles of the lower extremity will be indicated by weakness and a spastic-paretic gait. The patients make short, stumbling steps, scrape the feet upon the ground in walking, move the legs rigidly like stilts, and in consequence of contracture of the abductors push one thigh before the other with difficulty. At the same time the **knee-jerk** and **ankle-clonus** are greatly increased. **Cutaneous sensibility** and the **functions of the bladder** and the **rectum** exhibit no alteration. Should **bulbar symptoms** appear, the tongue undergoes atrophy, with the development of disturbances in the formation of the bolus and in articulation (hypoglossal paralysis). The muscles of the chin and the cheek undergo wasting and become rigid. The masseteric reflex is exaggerated. In consequence of vago-accessory and glossopharyngeal paralysis difficulty in swallowing, paralysis of the laryngeal muscles, and tachycardia occur. Inspiration-pneumonia and paralysis of respiration or of the heart are, therefore, frequent causes of death. The disorder generally terminates fatally in from one to three years. Recovery has been observed but rarely.

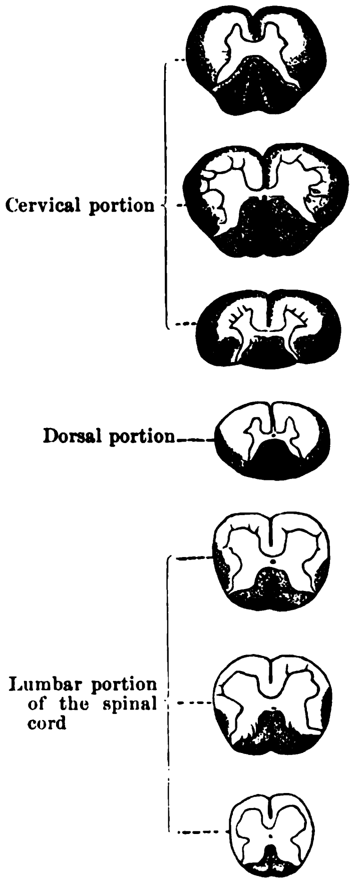

FIG. 82.—Anatomic alterations in the spinal cord from a case of hereditary ataxia: the diseased areas are shaded (after Friedreich and Schultze).

The **prognosis** is grave.

Diagnosis.—In the diagnosis care should, in the first place, be observed to avoid confusion with **spinal progressive muscular**

atrophy, although in cases of the latter, conversely from amyotrophic lateral sclerosis, the atrophy precedes the muscular paralysis, and the reflex movements are diminished or even abolished. Amyotrophic lateral sclerosis is generally distinguishable from **syringomyelia** from the presence of partial sensory loss and trophic disturbances in the latter. In the presence of **hypertrophic cervical pachymeningitis** irritative symptoms (pain) preponderate, and, in addition, other sensory disturbances occur and bulbar symptoms are wanting.

Treatment.—Good results are reported from the administration of **potassium iodid** (5.0 : 200—75 grains : 6½ fluidounces : 15 c.c.—1 tablespoonful thrice daily) and **sulphurous baths** (potassium sulphid, 150—5 ounces—to a full bath at a temperature of 28° R.—35° C.—95° F.).

Secondary Degeneration of Individual Columns of the Cord.—Secondary degeneration of the spinal cord is rather of anatomic than of clinical interest, so that a brief reference to the subject must suffice. The reason for its occurrence is found in the circumstance that, in accordance with the so-called law of Waller, the peripheral portions of nerve-fibers that are separated from their trophic centers undergo degeneration and atrophy. Three varieties of secondary degeneration of the spinal cord may be distinguished, namely, an *ascending*, a *descending*, and a *mixed* form.

Descending secondary degeneration of the spinal cord always develops when the corticomuscular or motor pyramidal tract is interrupted at any point in its course, so that the motor paths toward the peripheral side of the focus of disease are cut off from their trophic center, the motor pyramidal cells in the motor cortical center in the anterior and posterior central convolutions of the cerebrum. This condition occurs most commonly as the result of cerebral hemorrhage, which generally takes place into the internal capsule. The degenerated pyramidal tract may be visible in advanced cases to the naked eye in the middle portion of the foot of the related cerebral peduncle and in the pons. Within the pyramidal decussation of the medulla oblongata the greater portion of the degenerated [illegible] crosses over into the lateral crossed pyramidal tract of the opposite half of the spinal cord and only a small [illegible] same side and continues their course [illegible] pyramidal tract (p. 597, Fig. [illegible]). In the [illegible] of the spinal cord the area [illegible] the periphery of the [illegible] In the lumbar region [illegible] periphery of the cord [illegible] narrowing, as in

instance, in the accompanying illustration, while in other cases, on the contrary, it may be larger than the crossed pyramidal tract. Anatomically, there results degenerative atrophy of nerve-fibers, to which proliferation of the neuroglia becomes superadded. Further, even marked degeneration is frequently not distinguish-

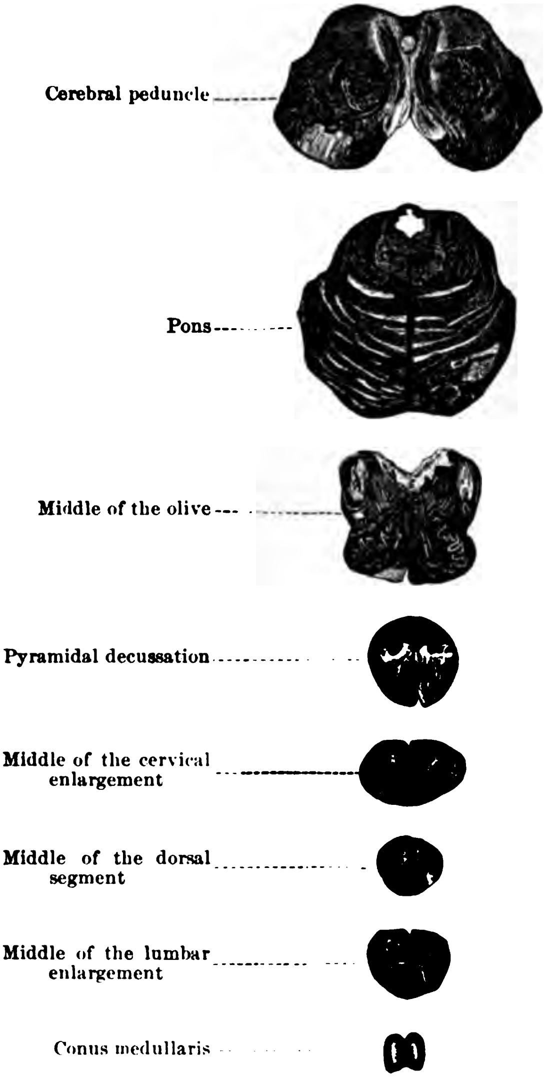
Cerebral peduncle
Pons
Middle of the olive
Pyramidal decussation
Middle of the cervical enlargement
Middle of the dorsal segment
Middle of the lumbar enlargement
Conus medullaris

FIG. 83.—Descending secondary degeneration of the right lateral pyramidal tract in consequence of hemorrhagic encephalomalacia of the left internal capsule; hardened in Müller's fluid; natural size (personal observation, Zurich clinic).

able in the fresh cord, although it at once becomes most distinct after hardening in Müller's fluid. Descending secondary degeneration of the spinal cord is beyond the possibility of diagnosis. In patients with this lesion *muscular contractures* and *exaggeration of the reflexes* are often present; but it has not been demonstrated that these phenomena are dependent upon the secondary degeneration in the cord. Both manifestations might be the result of long-continued paralysis, without etiologic relation to each other.

Ascending secondary degeneration of the spinal cord is in its pure form a rare disorder. It has been observed in isolated cases in association with neoplasms compressing the roots of the cauda equina. It has also been developed experimentally in animals by division of the posterior nerve-roots of the spinal cord. In the immediate neighborhood of the site of injury degeneration was found in the postero-external or columns of Burdach, but the degeneration extended upward into the postero-internal or columns of Goll, and it was observed that the degenerated nerve-fibers

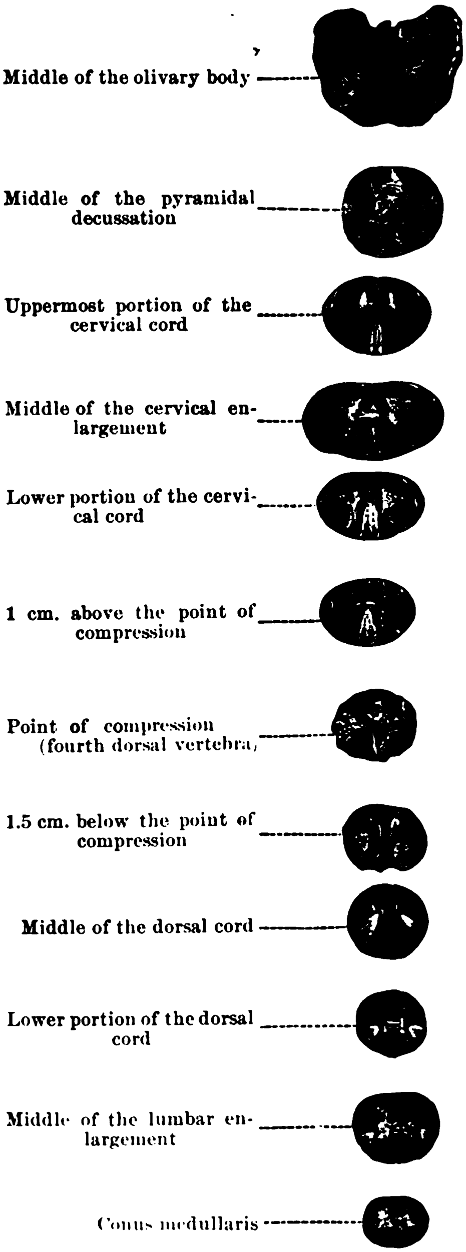
Middle of the olivary body
Middle of the pyramidal decussation
Uppermost portion of the cervical cord
Middle of the cervical enlargement
Lower portion of the cervical cord
1 cm. above the point of compression
Point of compression (fourth dorsal vertebra)
1.5 cm. below the point of compression
Middle of the dorsal cord
Lower portion of the dorsal cord
Middle of the lumbar enlargement
Conus medullaris

FIG. 84.—Compression-myelitis in a man, 62 years old, resulting from tuberculosis of the fourth dorsal vertebra; natural size; hardened in Müller's fluid (personal observation, Zurich clinic).

at progressively higher levels more closely approached the posterior median fissure and advanced forward.

Mixed secondary degeneration of the spinal cord develops after transverse lesions of the cord. Below the seat of disease the lateral and the anterior pyramidal tracts upon either side undergo degeneration. Above the transverse lesion, however, ascending degeneration takes place in the columns of Goll, in the lateral cerebellar tracts, and in the bundles of Gowers in front of the latter (p. 598, Fig. 84). As is well known, the intervertebral ganglia represent the trophic centers for the columns of Goll, and the columns of Clarke those for the lateral cerebellar tracts. As the columns of Clarke extend only down to the lower part of the dorsal cord, transverse lesions in the lumbar cord will not be followed by secondary degeneration of the lateral cerebellar tracts. Further, diffuse (traumatic) degeneration of the spinal cord will occur in the immediate neighborhood of the seat of disease, and the columnar degeneration will begin a slight distance beyond.

FUNCTIONAL DISORDERS OF THE SPINAL CORD OR NEUROSES OF THE SPINAL CORD.

ACUTE ASCENDING SPINAL PARALYSIS.

Symptoms, Prognosis, and Anatomic Alterations.— Acute ascending spinal paralysis, also known as Landry's paralysis, either sets in suddenly or is preceded by slight chill, febrile movement, pain in the back and in the extremities, and paresthesiæ as prodromes. The patient is conscious of weakness in one lower extremity or perhaps simultaneously in both, and the weakness soon gives place to marked flaccid paralysis. While at first only the muscles of the foot are involved, soon those of the leg and then those of the thigh become paralyzed, and then the paralysis extends to the abdominal, dorsal, and thoracic muscles, to the arms, and finally also to the tongue, the pharyngeal muscles, and the vago-accessory nerve, with the development of difficulty in breathing and accelerated heart-action. Other cerebral nerves (facial, abducens) are rarely involved in the paralysis. Occasionally death takes place within a few days as the result of inspiration-pneumonia or respiratory or cardiac paralysis, while in other instances it is deferred for several weeks.

The paralyzed muscles exhibit **unchanged electric irritability**, and waste, if at all, only after the paralysis has existed for some time (atrophy from disuse). The **bladder and the rectum** are not involved in the paralysis. **Sensory disturbances** are wanting or are prese[illegible] in insignificant degree. The **cutaneous** and **tendinous**

reflexes remain unaltered, or become gradually enfeebled after the disease has existed for some time. **Trophic alterations** are wanting, while **vasomotor disturbances** (discoloration of the skin, sweats) occur at times. **Fever** is not present, as a rule. **Consciousness** is preserved until death occurs. In numerous instances **enlargement of the spleen** and **albuminuria** were present, so that the appearance of an infectious disease was created. Unfortunately, the disease terminates fatally in the majority of cases. Improvement and recovery take place but seldom. It is distinctive of the disease that alterations in the nervous system capable of explaining the occurrence of the paralysis cannot be found.

Diagnosis.—The recognition of acute ascending spinal paralysis is not difficult if the typical symptoms are borne in mind. At times **acute ascending myelitis** simulates the disorder, but the former is generally attended with sensory disturbances, paralysis of the bladder and the rectum, and changes in the tendon-reflexes. **Acute ascending polyneuritis** also may give rise to confusion. It should be noted that in the presence of the latter disease degenerative electric reaction appears early in the paralyzed nerves and muscles, that the tendon-reflexes disappear, and that the muscles undergo degenerative atrophy within a short time.

Etiology.—**Exposure to cold** and **emotional disturbances**, but with greater propriety **antecedent infectious disease**, are named as causes of acute ascending spinal paralysis. The disorder is most common between the twentieth and the fortieth year of life, and in men.

Treatment.—Inunctions of **mercurial ointment** (5.0—75 grains—daily) have been made, particularly if syphilis had previously been present. No good is to be expected from **nervines.** The **galvanic current** also, applied to the vertebral column, would scarcely be capable of controlling the disease. Should paralysis of deglutition develop, it will be necessary to nourish the patient by means of the **stomach-tube.**

REFLEX PARALYSIS.

Reflex paralysis was looked upon until within recent years as a by no means rare occurrence, and it was observed particularly in the sequence of diseases of the female generative apparatus, the kidneys, and the bladder (urinary paraplegia), and the intestines (dysentery, intestinal worms). Its occurrence was attributed to paralysis of the functions of the spinal cord, in consequence of irritative conditions in various organs. It has, however, been shown recently that most cases of reflex paralysis are due to neuritis and myelitis that have developed by extension from the diseased organs. Nevertheless, reflex paralysis does appear to occur, and in support of this view it may be pointed out that some forms of paralysis disappear with surprising rapidity after removal of the fundamental disorder.

PSYCHIC PARALYSIS.

Psychic paralysis results from the influence of profound emotional disturbances, particularly fright, whence also the name of *fright-paralysis.* Paraplegia has been observed also as a result of *imagination.* Further, myelitic alterations may take place as a result of fright.

The **treatment** consists in encouraging suggestion and in making a mental impression on the patient.

DISEASES OF THE SPINAL MENINGES.

INFLAMMATION OF THE SPINAL DURA MATER (SPINAL PACHYMENINGITIS).

Inflammation of the spinal dura mater may involve either the outer or the inner aspect of this membrane, and accordingly a distinction is made between *external* and *internal spinal pachymeningitis.* Naturally, it may happen that an external pachymeningitis in particular may extend to the inner aspect of the dura, and thus give rise to internal pachymeningitis.

EXTERNAL SPINAL PACHYMENINGITIS.

Etiology.—External spinal pachymeningitis occurs most commonly as the result of **vertebral disease** (tuberculosis, carcinoma). At times inflammatory processes have invaded the vertebral canal through the intervertebral foramina or, after destruction of the vertebræ, through abnormal openings, and such a condition has been observed in connection with cheesy-tuberculous pleurisy, carcinoma of the esophagus, syphilitic ulceration of the pharynx, and deep bed-sores. It is doubtful if the disease may result from *exposure to cold.*

Anatomic Alterations.—The anatomic alterations vary with the causative factors. In some cases purulent, at times (in the presence of bed-sores) smeary and putrid, deposits are found upon the outer aspect of the spinal dura, beneath which the tissue of the dura is vividly reddened and swollen. At the same time accumulations of pus may have formed in the loose fatty connective tissue between the dura and the vertebral column (spinal peripachymeningitis). When the meningitis is tuberculous, cheesy deposits are not rarely found upon the spinal dura, and which may be several centimeters thick, and may exert considerable pressure upon the spinal cord and its roots. In cases pursuing a chronic course dense, cicatrix-like thickening occurs upon the outer aspect of the dura mater, and often also connective-tissue adhesions to surrounding structures.

Symptoms, Diagnosis, and Prognosis.—The disease is attended with **stiffness** and **pain in the vertebral column.** Pressure, percussion of the vertebral column, the passage of a sponge dipped in hot water, induce pain at the site of the inflammation. Not rarely complaint is made of **girdle-sense** in consequence of irritation of posterior nerve-roots. If spinal nerve-roots are compressed by inflammatory products to such a degree as to suffer loss of function, **paralysis** and **anesthesia** result. Often the clinical picture of **spinal pressure-paralysis** is developed.

In diagnosis the demonstration of spinal deformity is important, as this often indicates involvement of the dura. Nevertheless, the diagnosis is rather a matter of probability.

The **prognosis** varies with the nature of the primary disorder. Recovery is possible.

Treatment.—In the treatment **rest**, application of **ice**, and administration of **potassium iodid** are particularly deserving of consideration. Recently, **surgical treatment** has also been attempted (opening of the vertebral column and removal of cheesy masses).

INTERNAL SPINAL PACHYMENINGITIS.

Internal spinal pachymeningitis may appear in one of two forms: either as hypertrophic internal spinal pachymengitis or as hemorrhagic internal spinal pachymeningitis.

Hypertrophic internal spinal pachymeningitis is a rare disease, which has been attributed to *exposure to cold*, *syphilis*, and *alcoholic excess*. It is characterized by connective-tissue thickening of the dura, and not rarely also of the arachnoid and the pia, so that the spinal cord becomes surrounded by a thick band of connective tissue. The process is generally circumscribed, and is situated in the cervical portion of the cord with relative frequency, so that the condition has been designated cervical hypertrophic pachymeningitis. The dangers consist, in the first place, in irritation of the nerve-roots passing through the diseased tissue, and subsequently in paralysis. Besides, the clinical picture of spinal pressure-paralysis may be superadded in the presence of excessive compression of the spinal cord.

The patient complains principally of *stiffness* and *pain in the vertebral column*, and often also of a *constricting, girdle-like feeling* in the upper portion of the chest. In addition, there appear *neuralgia in the arms* and various forms of *paresthesiæ* (formication, burning). Also, *muscular twitching* is not rarely observed in the arms. At times *trophic disturbances* occur (thickening of the epidermis, formation of vesicles). All of these manifestations result from irritation of the nerve-roots. *Paralysis of the spinal* *nerve-roots* is attended with cutaneous anesthesia and loss of the reflexes, and generally gives rise first to paralysis of the muscles supplied by the median and ulnar nerves. The action of the extensors of the hand and the fingers therefore becomes predominant, and gives rise to contracture and fixation of the hand in dorsal flexion and to claw-fingers. The paralyzed muscles rapidly undergo atrophy and exhibit degenerative electric reaction. Finally, the clinical picture of *spinal pressure-paralysis* may be superadded. Paraplegia develops in the lower extremities, the tendon-reflexes become exaggerated, and paralysis of the bladder and the rectum occurs.

The disease often extends over years, and is at times susceptible of recovery. It is distinguished from *spinal progressive muscular atrophy* by the predominance of the sensory disturbances at the beginning of the disorder, a circumstance that is of importance also in the differentiation from *amyotrophic lateral sclerosis*, in which disease, besides, the reflexes are exaggerated. Therapeutically, employment may be made especially of potassium iodid internally and local applications of iodin and the ice-bag.

Hemorrhagic internal spinal pachymeningitis may occur, together with a similar process of the cerebral dura, particularly in the *insane* and in *alcoholics*, and is attended either with thin, vascular membranes that can be scraped off with a knife, or with blood-sacs of considerable size (hematomata), which upon section are found to consist of discrete collections of blood of varying age, separated from one another by membranes. Only hematomata at times give rise to symptoms by exerting pressure upon the spinal cord and the nerve-roots. Also, blood-vessels at times rupture suddenly in individual membranes, with profuse hemorrhage, which may compress and paralyze the spinal cord. The diagnosis cannot be made with certainty.

INFLAMMATION OF THE SOFT MEMBRANES OF THE SPINAL CORD (SPINAL MENINGITIS).

Inflammation of the soft membranes of the spinal cord involves the arachnoid, but especially the pia. *Arachnitis* and *piitis* are grouped together under the designation meningitis (leptomeningitis), and a distinction is made between *acute* and *chronic spinal meningitis*.

ACUTE INFLAMMATION OF THE SOFT MEMBRANES OF THE SPINAL CORD (ACUTE SPINAL MENINGITIS).

Anatomic Alterations.—Acute spinal meningitis is almost always *purulent in character*. The pia especially is infiltrated and covered with yellowish-green masses of pus, which are particularly abundant upon the posterior aspect of the spinal cord, because the patients generally occupy the dorsal decubitus. In this situation at times the pus envelops the entire spinal cord in a coherent membrane, while upon the anterior surface it is present in insular and disseminated areas. The pia and the arachnoid exhibit, besides, vascular **hyperemia**. The veins are greatly distended with blood, dilated, and markedly tortuous. Here and there vessels have ruptured, and generally small extravasations of blood have taken place. The **cerebrospinal fluid** is generally increased, and, besides, it is usually flocculent and turbid and slightly purulent. The **spinal cord** is in many cases unaltered; in others it appears hyperemic and soft, and at times it may even contain small abscesses. Purulent inflammation of the pia often extends to the *spinal nerve-roots.*

On *microscopic examination* the tissue of the pia and the arachnoid is found swollen and thickened, and containing innumerable round cells. The blood-vessels are greatly dilated and filled with red blood-corpuscles. Their walls are thickened and infiltrated with round cells. In preparations stained with aniline dyes bacteria can be discovered in places in recent cases. The *cerebrospinal fluid* contains large numbers of pus-corpuscles. These frequently form small masses admixed with fibrin, and correspond with the flakes of pus appreciable to the unaided eye. The *spinal cord* not rarely is wholly unaffected. At times nerve-fibers in proximity to the pia are inflamed and swollen. The inflammatory process may, however, invade the septa that penetrate the spinal cord from the pia. The *spinal nerve-roots* also are but slightly involved in the inflammatory process in some cases, while in others degeneration of nerve-fibers, round-cell accumulations, and vascular hyperemia may be encountered.

Etiology.—Purulent spinal meningitis occurs most commonly in association with purulent cerebral meningitis, and the condition is then designated purulent *cerebrospinal meningitis.* This affection occurs at times in epidemic distribution as an independent infectious disease, and will receive detailed description in the consideration of the infectious diseases. Here only those cases will be discussed in which the inflammation is confined to the soft membranes of the spinal cord. The affection under such conditions is often due also to *local causes* and to *extension from adjacent inflammatory processes.* The inflammatory irritant may enter the spinal cord from without, and therefore spinal meningitis is at times observed in association with deep **bed-sores** over the sacrum, with **suppuration in the pelvic connective tissue** as a result of puerperal septicemia, and at times also with **pleural empyema. Puncture of** or **injection into the dural sac** also is capable of exciting purulent spinal meningitis if unclean instruments or fluids containing bacteria or irritating substances are employed. In the same way operative measures, with opening of the vertebral canal, and **punctured** and **gunshot wounds** and **traumatism of other kinds** may give rise to acute spinal meningitis. At times the disease is dependent upon inflammatory processes in the **vertebræ** or **the spinal cord.**

Occasionally acute spinal meningitis develops in the course of **infectious diseases** (septicemia, pneumonia, erysipelas, typhoid fever, etc.). **Exposure to cold** also is said to be a cause, but probably it is only a contributory factor for the infection of the meninges.

In the majority of cases the *excitants of the inflammation* are bacteria, especially the Streptococcus pyogenes and the Staphylococcus pyogenes albus and aureus. The Meningococcus intracellularis has been demonstrated as the cause of epidemic cerebrospinal meningitis. Inflammation of the spinal meninges in consequence of purely chemic irritation is possible, but is probably rare.

Symptoms and Diagnosis.—*Local symptoms* are decisive in the recognition of spinal meningitis. The patient complains frequently at first of **stiffness** and **pain in the vertebral column**, and also pressure upon and percussion of the vertebræ, as well as movement of the vertebral column in sitting erect and in rotation of the body, excite pain. The occurrence of **rigidity of the neck** (*torticollis, wry-neck*) is of especial diagnostic importance, and is dependent upon irritation and contracture of the muscles of the neck. Under such circumstances the head may be strongly retracted, and it may be impossible to flex it upon the chest. On the other hand, rotation and dorsal flexion of the head are not interfered with. Often the muscles of the back also are in a state of permanent contracture, and the **vertebral column** appears curved with its convexity forward, so that the fist can be easily placed between the body and the bed. Contracture of the abdominal muscles causes **scaphoid retraction of the abdomen.** Often a sense of constriction about the trunk, and even a painful **girdle-sense,** may appear, and this is indicative of irritation of the posterior nerve-roots. Radiating **pains** in the lower extremities and **muscular twitching** also are symptoms of irritation of the nerve-roots. Generally there is **cutaneous hyperesthesia.** The irritability of the **vasomotor nerves** also is increased, so that even slight cutaneous irritation is sufficient to induce active and persistent reddening of the skin. *Irritative manifestations* are thus present throughout the entire clinical picture. Naturally, these may be followed by **paralytic phenomena** in the progress of the disease. *Muscular paralysis* develops; cutaneous hyperesthesia is replaced by *anesthesia; paralysis of the bladder and the rectum* appears; the exaggeration of the reflexes is followed by their *diminution* and *disappearance.* **Pupillary alterations** occur often, inequality of the pupils with especial frequency. Little diagnostic importance is to be attached to the *general manifestations.* The disease is almost always attended with *elevation of the bodily temperature,* although the intensity and the type of the fever are subject to no definite rule. The *duration of the disease* at times extends over only a few days, while in other instances the disorder is protracted over a few weeks, and gradually assumes a rather chronic course.

Prognosis.—The prognosis of acute spinal meningitis is grave. The principal danger consists in extension of the inflammatory process to the cerebral membranes, and death from increasing cerebral pressure or from paralysis of the vagus. Naturally, death may result, apart from these conditions, from progressive exhaustion, from paralysis of the bladder and urinary infection, or from septicemia in consequence of the fundamental disorder. At times muscular paralysis persists for a long time.

Treatment.—In the first place, the application of Chapman's **ice-bag** to the vertebral column is to be recommended in the treatment of acute spinal meningitis. Internally, **antipyrin** (1.0—15 grains—4 times daily) or **phenacetin** (1.0—15 grains—thrice daily) may be prescribed for the relief of the pain. If this be intense, **subcutaneous injections of morphin** may be necessary. Scarcely any therapeutic effect will be yielded by **lumbar puncture**. The patient should be placed at **rest in bed**, and receive only liquid diet, preferably a **milk-diet**.

CHRONIC INFLAMMATION OF THE SOFT MEMBRANES OF THE SPINAL CORD (CHRONIC SPINAL MENINGITIS).

Anatomic Alterations.—Chronic spinal meningitis is attended with **connective-tissue thickening** of the pia and the arachnoid. Often **connective-tissue adhesions** have formed between the pia, the arachnoid, and the dura. The morbid process usually occurs in circumscribed foci, is most markedly developed upon the posterior aspect of the spinal cord, and involves with especial frequency the lumbar cord, and least commonly the cervical cord. At times the pia particularly presents a brownish or blackish discoloration, indicative of antecedent hemorrhage. Calcareous plates not rarely form, particularly in the arachnoid columns. The **cerebrospinal fluid** is either unchanged or increased in amount. At times it appears slightly turbid. On microscopic examination of the **spinal cord** the unusually marked development of the pial septa not rarely attracts attention. Often nerve-fibers in the neighborhood of the pia are degenerated, and at times there is complete marginal degeneration of the white substance of the spinal cord. The **spinal nerve-roots** also often exhibit thickening of the pia, and on microscopic examination present connective-tissue hyperplasia and nerve-degeneration.

Etiology.—In the etiology of chronic spinal meningitis the same causative factors are operative as in that of acute meningitis (pp. 604 and 605). At the same time it may happen that acute inflammation occurs at first, which slowly passes into the chronic form, or, in other instances, chronic spinal meningitis develops independently from the outset. *Syphilis, alcoholism,* and *chronic diseases of the spinal cord* (chronic myelitis, tabes dorsalis) particularly are considered especially common causes of chronic spinal meningitis.

Symptoms and Diagnosis.—The symptoms of chronic spinal meningitis are identical with those of acute meningitis, except for the absence of fever and the duration of the disease often for several years. *Irritative symptoms* are present at the beginning, and consist in *stiffness* and *pain in the vertebral column, rigidity of the neck,* a *girdle-sense, radiating pains in the lower extremities, paresthesiæ, muscular twitching and contractures, cutaneous hyperesthesia,* and *exaggeration of the reflexes.* After a time *paralysis* develops if the spinal cord, and particularly the nerve-roots, are degenerated. The *muscles* are rather paretic than paralytic, and undergo degenerative atrophy and exhibit degenerative electric reaction. The *reflexes* become enfeebled or disappear. *Cutaneous anesthesia* develops, and frequently *paralysis of the bladder and the rectum* occurs, with the development of urinary infection, bed-sores, and septicemia. At times the disorder assumes an acute character. By extension of the inflammatory process to the meninges of the brain and the medulla oblongata death may result from excessive cerebral pressure or from respiratory or cardiac paralysis.

Prognosis.—Recovery from chronic spinal meningitis is not impossible. Nevertheless, the prognosis is serious, particularly in the presence of paralysis of the bladder and the rectum.

Treatment.—**Causal treatment** is indicated when there is a history of alcoholism or of syphilis. In the case of drunkards alcohol should be withdrawn, and syphilitics should be treated with inunctions of mercurial ointment and the internal administration of potassium iodid. *Symptomatically* the use of **warm baths** (28° R.—35° C.—95° F.) should be recommended. **Saline baths** also may be employed. **Mercurial inunctions** and potassium iodid also have been employed in non-syphilitic cases, but yield no definite results. When muscular paralysis develops resort should be had to **massage** and **electricity**.

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Etiology.—Hemorrhage into the membranes of the spinal cord is only of subordinate clinical significance. It occurs at times as the result of **traumatism**, and not alone after punctured, incised and gunshot wounds and fractures and luxations of the vertebræ, but also after concussion, powerful expulsive efforts, and heavy lifting. It is often a sequel of **circulatory disturbances**. It occurs particularly in conjunction with convulsive disorders (eclampsia, epilepsy, tetanus). Chronic diseases of the heart and of the respiratory organs also at times cause meningeal hemorrhage, which occasionally accompanies inflammation of the meninges, the spinal cord, or the vertebral column. Not rarely meningeal hemorrhage occurs in the course of **infectious diseases**, apparently because the vessels become abnormally permeable to the blood-corpuscles as the result of the infective process. Sometimes blood passes from the intermeningeal spaces of the brain into the corresponding spaces of the spinal cord. It may also happen that an **aortic aneurysm** ruptures into the vertebral canal and pours its contents into the intermeningeal space. A similar result has been observed also from **rupture of the vertebral artery and the spinal artery.** At times it is not possible to discover a cause for the condition—*cryptogenetic meningeal hemorrhage.*

Anatomic Alterations.—Meningeal hemorrhage can be readily recognized, and in accordance with its age presents a bright-red or a brownish-red appearance. In size it varies from punctate and barely visible spots to extravasations that may extend throughout the entire length of the spinal cord. Should absorption take place, brownish pigment-spots, subsequently becoming black, often persist. In accordance with the seat of the hemorrhage several varieties are distinguished. *Epidural hemorrhage* occurs upon the outer aspect of the dura, particularly in the fatty connective tissue that separates the dura from the inner aspect of the vertebral canal. *Subdural hemorrhage* (formerly designated also arachnoid) occurs in the narrow, capillary space between the dura and the arachnoid. In this category belongs the *internal hemorrhagic pachymeningitis* described on p. 603. *Subarachnoid hemorrhage* is not rarely the result of extravasation of blood into the corresponding portion of the brain, and together with cerebrospinal fluid may be present in the interstices of the arachnoid. *Subpial hemorrhage* can be conceived as resulting only from displacement of the pia from the structure of the cord by the blood.

Symptoms and Diagnosis.—The **sudden onset** and the **predominance of irritative symptoms** are distinctive of meningeal hemorrhage. Naturally, these features can be expected only if the extravasation of blood is sufficiently large to cause irritation of the meninges and the spinal nerve-roots. Hemorrhages of smaller amount may be unattended with symptoms and fail of recognition during life. At times a sense of drawing and other unusual sensations in the vertebral column have preceded the occurrence of the hemorrhage for a day or even longer, as *prodromes.* The occurrence of a hemorrhage of considerable amount is attended with **severe pain in the vertebral column,** whose seat corresponds with that of the hemorrhage, and is probably dependent upon the sudden distention of the meninges. The patient is conscious of **stiffness** and generally of increase in the pain on movement of the vertebral column. Should the hemorrhage be seated in the cervical cord, **rigidity of the neck** may develop. Irritation of posterior nerve-roots may cause a **girdle-sense** about the trunk, and often also radiating pains in the lower extremities. **Paresthesiæ, hyperesthesia, muscular twitching** and **contractures** are not rarely present. Hemorrhage of sufficient extent is capable of causing paralysis of spinal nerve-roots and the spinal cord by compression. Under such circumstances **cutaneous anesthesia, muscular paralysis with degenerative atrophy and degenerative electric reaction, enfeeblement or abolition of reflex movement,** and **paralysis of the bladder and the rectum** may develop. The distribution of the manifestations described depends upon the seat and the extent of the hemorrhage. In contradistinction from spinal hemorrhage, meningeal hemorrhage is distinguished by the presence of irritative symptoms at the beginning.

Prognosis.—The prognosis should be guarded. Danger resides in the fact that hemorrhage at a high level may injure the nerve-roots in the medulla oblongata. *Bulbar symptoms* (disturbance in articulation and in deglutition) develop, and in connection with which paralysis of the vago-accessory nerve may give rise to a fatal termination through paralysis of respiration or of the heart. At times meningeal hemorrhage may be complicated by *meningitis*, with its serious dangers. Should paralysis of the bladder and the rectum develop, the danger of *urinary infection, bed-sores,* and *septicemia* arises. At times *paralysis* remains permanently. Complete recovery is, however, by no means impossible.

Treatment.—The application of a Chapman **ice-bag** to the vertebral column should be directed; a diet, principally of **milk**, be prescribed; the use of coffee, wine, tea, and alcoholics be interdicted; and free, easy **evacuation of the bowels** be provided for. Severe pain should be relieved by phenacetin (1.0—15 grains—thrice daily), antipyrin (1.0—15 grains—four times daily), or subcutaneous injection of morphin. Persistent paralysis will require **massage**, which is to be preferred to the application of **electricity**. After the disappearance of all irritative symptoms **baths** may be of advantage, particularly saline baths.

NEOPLASMS OF THE SPINAL MENINGES.

Anatomic Alterations.—Neoplasms of the spinal meninges arise, as a rule, from the **dura mater**, and generally are spherical in shape. They vary in size, although they rarely exceed 5 cm. in length. Fibromata, lipomata, myomata, sarcomata, psammomata, melanomata, neuromata, tuberculomata, gummata, enchondromata, osteomata, carcinomata, and lymphangiomata have been observed. By reason of the small size of the vertebral canal, extensive development of the neoplasm is possible only by penetration of the new-growth through the intervertebral foramina and proliferation externally, or by compression of the spinal cord and the nerve-roots. At times neoplasms have formed a deep excavation in the spinal cord, while in other instances the cord and adjacent nerve-roots are flattened. As a result of the compression softening of the spinal cord may readily result. Below and above the point of compression secondary degeneration occurs after a time: above in the column of Goll, the lateral cerebellar tracts, and the bundle of Gowers, and below in the lateral pyramidal tracts.

Etiology.—**Syphilis** is definitely known as the cause for gummata, and **tuberculosis** for tuberculomata. Otherwise, nothing is known with regard to the etiology. **Traumatism, pregnancy,** and the **puerperium** are further included among the causative conditions. Malignant neoplasms (sarcoma, carcinoma) at times arise by **metastasis** from antecedent neoplasms in other organs. Experience has shown that *men beyond the fortieth year of life* are most commonly attacked.

Symptoms and Diagnosis.—Neoplasms of the spinal meninges give rise to morbid manifestations only if at first they irritate the membranes, and subsequently cause paralysis as a result of excessive compression of the spinal cord or the spinal nerve-roots. The **irritative symptoms** are the same as those that have been mentioned in previous sections: Pain and a sense of stiffness in the vertebral column, a sense of constriction, shooting pains in the extremities, hyperesthesia, paresthesia, muscular twitching and contracture. In the presence of **spinal pressure-paralysis** the clinical picture, in accordance with the existing conditions, may be that of total transverse lesion of the spinal cord (paraplegia, para-anesthesia, exaggeration of the reflexes, paralysis of the bladder and the rectum), or that of a unilateral lesion of the cord, or a circumscribed injury of the cord. In the last event motor or sensory paralysis will predominate accordingly as the tumor compresses the spinal cord from in front or behind. **Compression of the nerve-roots** also will cause paralysis of motility and sensibility. It is distinctive for the paralyzed muscles to undergo rapid degenerative atrophy, with degenerative electric reaction and abolition of reflex movement.

The *duration of meningeal neoplasms* extends at times over several years. The dangers consist in a complicating meningitis, in excessive exhaustion, in the presence of paralysis of the bladder and the rectum, in cystitis and urinary infection, and in bed-sores and septicemia.

The **diagnosis** of neoplasms of the spinal meninges is attended with unusually great difficulty. There is generally a gradual development of the symptom-complex of meningeal irritation. The presence of neoplasms in other organs is a matter of great importance, thus rendering probable the existence of metastatic new-growths. In contradistinction from tumors of the spinal cord itself, irritative symptoms predominate in the presence of spinal neoplasms. The *nature of the neoplasm* is generally involved in doubt, except in the case of metastatic growths. With regard to the *seat of the disease*, local changes in the vertebral column and the distribution of the symptoms are decisive. Tumors of the cauda equina not rarely are attended with circumscribed cutaneous anesthesia of the perineum, the scrotum, the penis, and the posterior aspect of the thigh. In addition, paralysis of various muscles and the bladder and the rectum, and abolition of reflex movement, occur.

Prognosis and Treatment.—Although meningeal tumors have recently been successfully removed by **surgical means** after opening the vertebral column, the prognosis is nevertheless serious in the majority of cases. **Causal therapy** should be employed in cases of syphilis (mercurial inunctions, internal administration of potassium iodid).

In a few rare cases *cysticerci* and *echinococci* have been found in the spinal meninges, and the latter have been removed by operation.

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